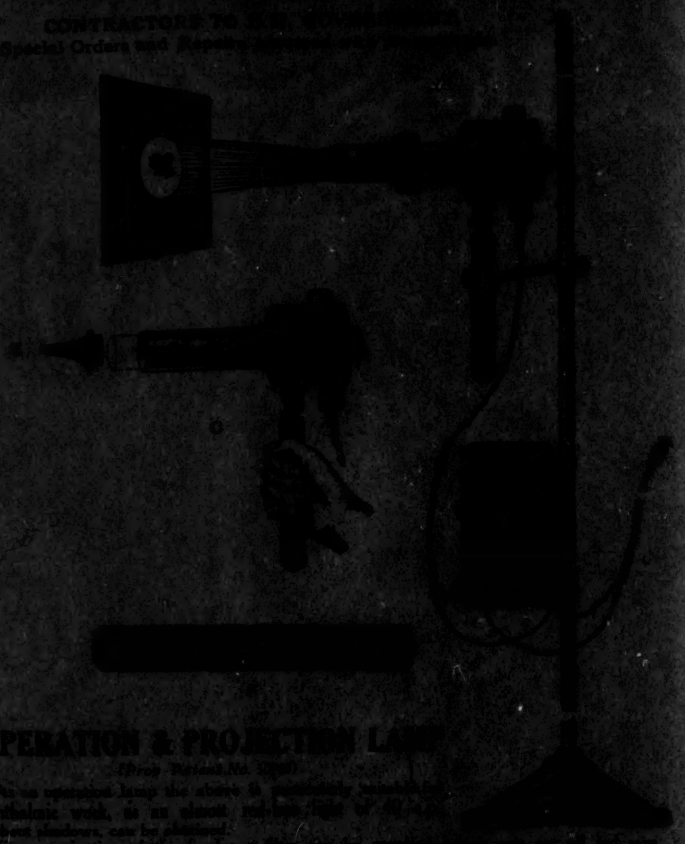

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COMMUNICATIONS

PATHOGENESIS OF MICROPHTHALMIA

BY

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INDIA

Case No. 1 (By kind permission of Mr. Juler)

History.—L.J., a female child, aged four days, out-patient Royal London Ophthalmic Hospital. Mother, aged 23 years, unmarried and this is her first baby. No history of any miscarriage, delivery was quite natural, no instruments used. Mother quite healthy. There is no history of any inflammation of the eyes since birth.

Grandmother had ten children of whom five are alive, one child was born with congenital contraction of fingers and toes on both sides. He had no nose but two tiny holes instead. The eyes, as far as could be ascertained, were quite normal.

The alleged father in the present case is described as a big, tall and handsome boy, motor driver by trade.

Examination.—The baby looks quite normal and there is no other abnormality present. The orbits are well formed, but the eyelids are on the small side. Lid retractors are necessary for a thorough examination on account of entropion and spasm of the lids.

There appears to be no development of the eyeball on either side. The sockets are lined by conjunctiva all through, the conjunctival sacs are well formed but nothing in the shape of nodules, etc., is detected by the fingers.

On the left side in the centre of the conjunctival sac is a small depression at the bottom round which there is some slightly pigmented scar tissue. This seems to be the only representative of the eyeball present, and, on histological examination it would probably prove to be the posterior retinal epithelial layer, thus illustrating the lowest form of microphthalmia. I do not mean to discuss anophthalmia in this paper.

Case No. 2 (By kind permission of Mr. Doyne)

B.W., a female child, aged 14 days, was brought to the Hospital for Sick Children, Great Ormond Street, on January 9, 1925, with a deformity of the right eye.

History.—There is no history of any deformity in the parents, mother has three children, all girls, the eldest being five years. The other two are perfectly healthy. The present child was born at full time, confinement being quite normal. There is no history of any inflammation of the eyes at birth.

Examination.—The child is very poorly developed and inclines her head to the right side. There are four pedunculated dermoids present; three are well shown in the photographs, the fourth being situated in the right nostril. In addition there is an accessory auricle on the right side (not shown in the photograph). The right lower lid is bulged out by a plum-coloured swelling, soft in consistence, and the size of a large marble. This swelling was noticed at birth. The upper lid appears to be quite normal, and the conjunctival sac is well formed, but there is nothing to be felt in the shape of an eye. The left eye is apparently quite normal.

The fluid drawn from this cyst was unfortunately contaminated with blood. The pathological report is as follows:

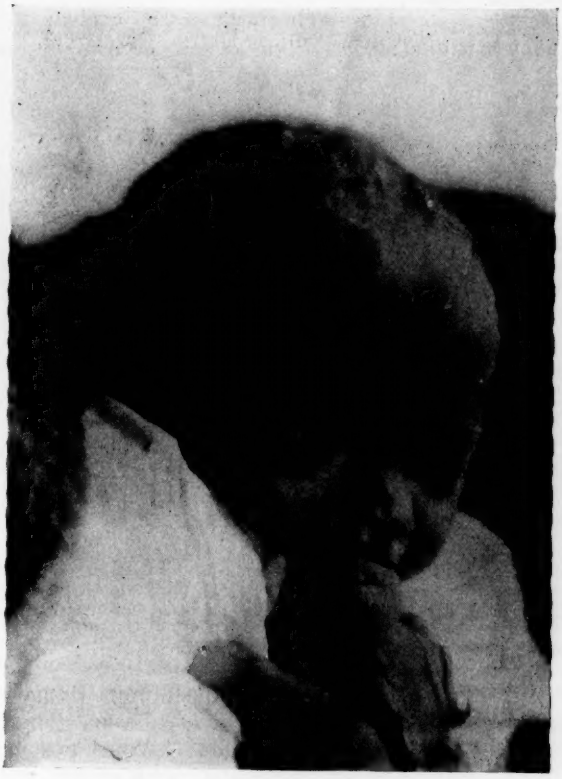
Numerous red blood corpuscles. Some polymorphs and mononuclear cells. Cultures sterile.

The swelling on the lower lid was dissected out by the following operation:

First of all the outer canthus was slit open to allow eversion of the lower lid. A horizontal incision was made on the conjunctival aspect along the margin of the lid. The conjunctiva thus severed was dissected backwards and the swellings in the lid removed. Lying to the right, and posterior to the cyst, was found a dermoid which was accidentally punctured, a quantity of greenish fluid escaping from it. The retinal cyst itself was found to be attached to a small hard mass of tissue, a microphthalmic eye.

The eye along with the retinal cyst was divided vertically. On section the microphthalmic eye consists of a mass of fibro-muscular tissue in the centre of which is a cavity containing uveal pigment. In shape it resembles a round disc except at its postero-inferior

border where the optic nerve enters it. Here there is a *slight protrusion.* In the section the optic nerve has been divided longitudinally and is traceable right on to the cavity of the eyeball. In the cavity which is situated slightly to the posterior



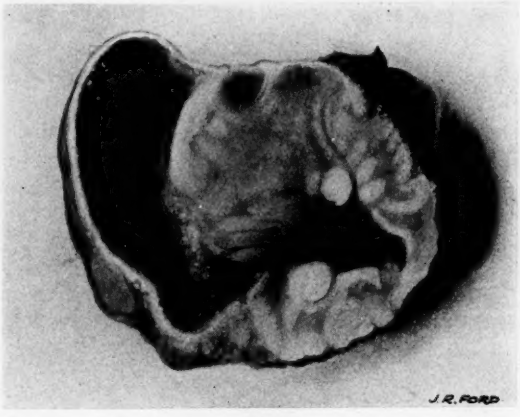
CASE NO. 2.

Note bulging of the right lower lid due to retro-palpebral retinal cyst and two dermoids along the line of naso-orbital suture.

aspect of the centre of the eyeball is the uveal pigment surrounded by darkish retinal elements. Lying to the infero-medial surface and reaching the anterior end of the eyeball is the retinal cyst. It is the shape of a half moon. Its communication with the cavity of the microphthalmic eye is situated at its postero-superior end, *i.e.,* just anterior to the entrance of the optic nerve into the cavity of

the eyeball. The cyst wall is about 1.5 millimetres in thickness, and is lined by a brownish membrane which is probably a retinal layer. Its outer covering looks like fibrous tissue. Celloidin sections of the microphthalmic eye and the retinal cyst were cut serially and stained, some with haematoxylin and eosin, others with Weigert's stain.

Microscopic description.—The rudimentary eyeball consists of a round mass whose outer portion is composed of fibrous tissue

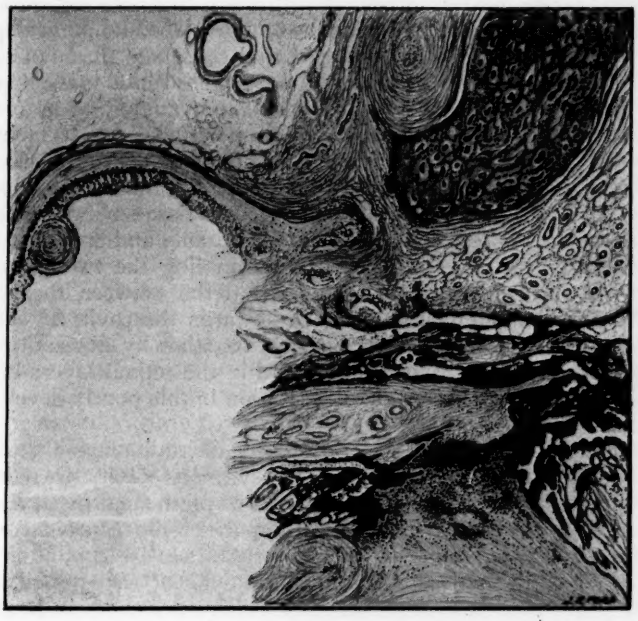


CASE NO. 2.

Note the microphthalmic eye with an orbital cyst on its inferior surface. The optic nerve is seen entering the posterior border of the eye. Its cavity is full of uveal pigment and retinal elements, which are seen protruding into the cavity of the cyst through a gap at the posterior end of the choroidal fissure.

lamellae arranged irregularly. In the fibrous tissue mass are situated some muscular bundles and areolar tissue. The thickness of this fibrous tissue is very variable. At its postero-inferior border is the entrance of the rudimentary optic nerve. It traverses the whole thickness of the fibrous tissue layer. Between the optic nerve and the fibrous tissue is a space continued right forward to where the nerve enters the interior of the eyeball. Here the fibrous tissue surrounding the nerve touches its sides. This is the vaginal space of the optic nerve. The nerve bulges out in the cavity of the eyeball, thus resembling the optic papilla, across which fibrous tissue lamellae stretch from one side to the other, so cutting off any communication between the nerve and optic cavity. The proper sheath of the optic nerve is composed of a thin layer of fibrous tissue. The nerve itself shows the typical appearance,

i.e., neuroglial bundles separated by fibrous tissue septa. Inferiorly and external to the fibrous tissue surrounding the nerve, a few millimetres from the entrance of the nerve, between it and the superior aspect of the posterior end of the retinal cyst, are two fairly large blood-vessels cut across. Further forwards and nearer



CASE NO. 2.

Only a part of the slide is drawn. It shows the communication between the cavity of the eyeball and the cyst; optic nerve; the cyst wall and blood vessels lying between the postero-superior aspect of the cyst; and the inferior surface of optic nerve.

the nerve are two smaller vessels. I take these to be the central artery and vein of the retina arising from their parent trunk. These blood-vessels are buried in fat and loose areolar tissue. Though in none of the slides can I make out the artery or the vein actually entering the substance of the nerve, I have no doubt in my mind that if antero-posterior sections were cut this would be apparent.

The cavity of the eyeball is full of uveal pigment and fibrous tissue. The latter in this situation is much more cellular than that representing the sclera, and is mainly situated outside the finger-

like processes of uveal pigmented structure. Imbedded in the fibrous tissue forming the outer covering of the microphthalmic eye are bundles of muscle tissue and a piece of cartilage. Situated on the infero-medial side of this rudimentary eyeball is the retinal cyst. As stated in the macroscopic description, its communication with the interior of the eyeball is situated at the superior aspect of its posterior border. Prolonged through this opening into the cavity of the cyst are the contents of the globe, *i.e.*, uveal pigmented structure, some retinal epithelial cells, and fibrous tissue. The retinal cyst itself is composed of an outer fibrous tissue layer and an inner layer that resembles the two granular layers of the fully developed retina. Between the outer fibrous and the inner retinal epithelial layer is a variable amount of neuroglial tissue.

Under the high power of the microscope the inner layer is seen to be divided into two, with loose areolar tissue between them. These two layers rest on a basement membrane, and are composed of retinal epithelial cells somewhat resembling the two granular layers of fully developed retina. Scattered between these two layers are globular masses bulging towards the cavity. These consist of embryonic retinal cells held together by fibrous tissue. In some slides cystic spaces are found in the retinal layers of the cyst. These represent cystic degeneration in this poorly developed retina.

Summary.—We find in this case a rudimentary eyeball associated with a retinal cyst. The vitreous cavity is reduced to a mere slit situated between the tufts of pigmented uveal tissue. There is a gap left at the posterior part of the choroidal cleft, through which the cavity of the microphthalmic eye communicates with that of the retinal cyst. The contents of the eyeball, *i.e.*, uveal pigmented structure and retinal elements, are seen protruding through this gap into the retinal cyst. The uveal structures are not prolonged along the cyst wall, but the retinal elements line the cavity.

At this stage it is advisable to recapitulate some of the important points in the development of the eye.

Two hollow diverticula bud out from the lateral aspects of the forebrain. These, after the closure of the anterior neuropore, are called the optic vesicles. The peripheral part of each expands to form the optic bulb, while the proximal part gives rise to the optic stalk; growth in both these parts continues and this results in the invagination of the optic bulb to form the optic cup in which is received the lens vesicle arising as a thickening of the ectoderm overlying the optic bulb. The invagination of the optic bulb does not cease at its lateral end but is continued right on to its inferior surface and reaches the optic stalk. Through this, the so-called choroidal fissure, mesoblastic tissue makes its way into the optic

bulb and stalk; in the latter position an artery is developed (arteria centralis retinae).

Of course the whole of the optic elements at this stage are surrounded by mesoderm. We need not consider here what happens to this, but the fate of that portion of it which is inside the optic bulb and that which fills up the choroidal fissure, *i.e.*, between the two lips of the groove situated on the inferior surface of the optic bulb, does concern us. The former gives rise to the vitreous body. The mesoderm that enters along with the lens vesicle has nothing to do with the formation of the vitreous body, as proved by Hermann Becker[1]. In his case the eye was removed from the body of a child five months old. It had increased in size since the baby was one month. On histological examination there was absence of the lens, pupillary opening, iris, ciliary body, and anterior chamber. A choroido-retinal coloboma with thinning of the sclerotic was present. The condition of the retina and pigment showed that the primary vesicle had developed into the secondary, but its inversion had not proceeded in the normal manner. Becker believes that no lens was formed in this case. He cannot decide with absolute certainty as to whether the introversion of the primary optic vesicle ever took place. Vitreous existed though no lens was ever formed. This mesoblastic origin of vitreous explains the occurrence of fat, cartilaginous tissue, and bone in the atypically developed vitreous. As the vitreous body increases in size ample room is afforded for the developing retina to spread out. The plug of mesoderm between the two lips of the choroidal fissure decreases in size *pari passu* with growth of the retinal layers. Ultimately the two retinal layers fuse with their fellows of the opposite side, thus cutting off any connection between the mesoderm that forms the vitreous body and that which surrounds the optic elements and forms the sclerotic, etc. The site where the fusion of the two retinal layers takes place is a weak spot, and if by any means the tension in the vitreous cavity of the developing eye increases, this gives way, with the result that the contents of the globe escape into the surrounding mesoderm and form a retinal cyst. It is quite conceivable that the degree of vascularity of the plug of mesoderm between the two lips of the choroidal fissure influences its fate. If it is very vascular the retinal layers fail to suffocate it by their growth, and instead of fusing with their fellows they curl over. This has been proved histologically by van Duyse[2], who demonstrated four retinal layers at the neck of a retinal cyst. van Duyse, Junior[3], describing a case of true coloboma of the rudimentary optic nerve, aplasia of their retro-ocular segments gliosis of colobomatous cyst, and entropion of the border of iris in a rabbit one year old says: "In one eye on account of the

minimal development of the vitreous the small space devoted to the development of the retina has pushed that structure outside; it came in contact with the invaginated mesoderm and is evaginated and herniated. Its external expansion at the entrance of the rudimentary optic nerve on the infero-nasal side, has caused the formation of a colobomatous retro-ocular cyst."

The optic stalk of course gives rise to the optic nerve. This is brought about by obliteration of its cavity and the growth in it of nerve fibres most of which are centripetal and are the axons of the nerve cells of the ganglionic layer of the retina.

This case illustrates microphthalmia at a more advanced stage than Case No. 1.

These two cases illustrate arrest of development of the eyeball at a very early stage and I mean to discuss them before I describe the other two, in which arrest of development, or aberrant development as the case may be, has taken place much later.

Case No. 1.—This case illustrates the lowest type of microphthalmia and I am unable to find in the literature of this subject, any other recorded case that resembles the present one. In the absence of histological examination it is very difficult to say at which stage the development has been arrested but the appearance of the depression with the pigmentation all round, strongly suggests that the invagination of the primary optic vesicle has occurred and that the pigmented structure is really its posterior layer laden with pigment granules. What happened to the retina proper, that is the structure arising from the anterior layer of the primary optic vesicle, is very difficult to say.

Case No. 2.—This case illustrates *par excellence* microphthalmia associated with an orbital cyst, an affection not very infrequent.

In this case the development has proceeded quite normally up to the stage of the formation of the vitreous body. This structure arises from the mesoderm that is invaginated through the choroidal fissure which is situated on the inferior surface of the optic vesicle. The amount of vitreous depends on the consistence and vascularity of the intruded mesoderm (Carl Hess)[4]. If this is thick very little vitreous is formed with the result that the developing retina cannot get space to spread, and consequently gets folded, and thus the tension inside the globe is increased with the result that a few folds of retina are extruded through the choroidal fissure, this being the weakest spot, and ultimately give rise to these orbital retinal cysts. This is what has happened in this case. The usual position for these retinal cysts is the middle of the lower lid or the lower part of the orbit, but according to van Duyse[5] they are occasionally retro-ocular. He describes a case of microphthalmia with an orbital cyst which had two compartments, retro- and sub-ocular and an extra-orbital retro-palpebral.

Rindfleisch[6] who examined a microphthalmic eye associated with a retinal cyst from a foetus six to seven months old, which was hydrocephalic, and consequently the orbital roof was pressed down so that it was convex below, came to the conclusion that alterations in the orbital roof compressed the globe so bringing about a re-opening and widening of the choroidal cleft and an extrusion of the retinal tissue into the surrounding mesoblast.

This peculiarity was also noticed by van Duyse[7] in a new-born child 12 months old who had dolichocephalic hydrocephalus with an antero-posterior diameter of 17.5 centimetres and 14 centimetres transversely. The left frontal boss projected strongly in front of the glabella, the two frontal bones were separated, and there was double hare-lip and cleft palate. The child had talipes equino-varus on both sides, six fingers and a rudimentary penis. He goes on to say that in the actual subject the ventricular pressure had depressed the orbital roofs which were flat; this peculiarity was also noticed in the first child of the same parents. It is such as to exert an influence on the shape of the bulb so as to diminish its sagittal diameter.

The three cases quoted above would show that in some cases an alteration in the shape of the orbital roof presses the globe downwards, thus causing an increased intraocular pressure that opens up the defectively closed choroidal fissure and pushes the fold of the retina, which gives rise to these cysts, out; but this alteration in the orbital roof is not always found, so it cannot explain all the cases.

Kundrat[8] looks to a defect in development of the middle cerebral vesicle as the cause of the malformations which characterize these cysts, and the microphthalmia which invariably accompanies them, and in several of his cases gross deformities in the brain structure were discovered. But there are many cases of microphthalmia on record where there is no defect in the brain and *vice versa*.

The walls of these cysts are usually composed of two coats, an outer one of fibrous tissue continuous with the sclerotic and an inner of more or less highly developed retina. In a case examined by Treacher Collins[9] the inner coat was represented by bodies normally found in the granular layers of the retina, arranged in separate patches. In another case examined by the same author in which there were two cysts, these were formed by a continuous layer of branching cells and granular bodies. In van Duyse's case, described above, in all sections the internal layer lining the cystic cavity with the exception of nodules of local gliosis proved itself to be retina incompletely differentiated and inverted. The neck of the cyst was lined by folded retina (in four layers). He goes on to say that he described the case (brother of the present one) with

microphthalmia and retro-palpebral cysts on both sides. These cysts communicated with the interior of the eye and had the same lining as the above. Rindfleisch found an inner wall of the cyst composed of retina with its layers well developed, with pigmented epithelium, and an elastic lamina. The inner surface of the retina was directed towards the interior of the cysts.

I have shown that these retinal cysts are due to the extruded folds that lie in the mesoderm, having been surrounded by it, becoming shut off, and then going on to cyst formation. After being in this way shut off from the body of the bulb, active growth in the separated portion does not cease, but, up to a certain point, proliferation of the elements proceeds, with the result that the retinal elements more or less fully formed are found lining these cysts. This is what we find in the case under discussion.

Case No. 3.—The microphthalmic eyes to be described were removed from a new-born pig. There is no record of any other abnormality in the animal save the other eye which is also microphthalmic, and will be described as the next case. Both these eyes were sent to the Pathological Department of the Royal London Ophthalmic Hospital by Sir John Parsons, and I have to thank Sir John and Mr. Humphrey Neame for their kind permission to study and report these rare abnormalities.

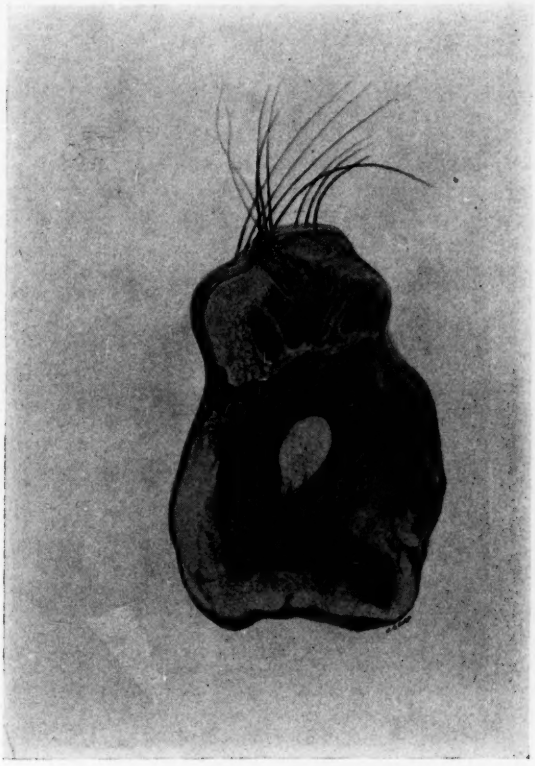
There is no record as to which side the eyes belong so I am describing them as "A" and "B."

The antero-posterior length of the first specimen "A" is 13 millimetres and it is 8 millimetres in thickness. In contour it is markedly irregular. At the posterior pole there is a cord-like structure leaving the globe, which on first sight looks very much like the optic nerve. This is well shown in the diagram which represents one-half of the eye. Of the other half microscopic sections were cut. As shown in the figure, the cornea is replaced by a hairy dermoid, which is encroaching on the limbus on both sides. In the cavity of the dermoid cyst are numerous sebaceous glands and remains of fatty tissue. In the centre of the cavity of the globe is a white glistening mass of cartilage. The rest of the cavity is full of a dark structure which seems to be folded up embryonic retinal epithelium, mixed up with choroid and remnants of vitreous body. There is just a trace of lens seen anterior to the cartilaginous piece.

The sclerotic coat is continuous all round and there is no ectasia or cyst formation discoverable. In this specimen there is no sign of any coloboma of the choroid, etc.

Microscopic description.—The half eye was imbedded in celloidin and horizontal sections were cut and stained with haematoxylin and eosin.

The normal cornea is replaced by a dermoid growth which encroaches on the limbus on the left hand side in the slides. The corneal epithelium is replaced by a pavement epithelium, many layers thick, with numerous papillae which resemble those of the



CASE NO. 3.

Note corneal dermoid with fibrous tissue cord at the posterior end, also a piece of hyaline cartilage and dark structure in the cavity of the globe (epithelial retinal tissue).

skin. The hair follicles are seen surrounded by sebaceous glands, some containing hair roots. Next to the epithelium the cyst wall is composed of fibrous bundles arranged irregularly: the separate fibres are but poorly supplied with nuclei. The cyst wall that is interposed between the ocular cavity and the cyst itself, is only one-third of the thickness of the rest of the wall, and, of course,

has no epithelial covering. It resembles sclerotic in structure, and is different from the rest of the cyst wall. The cyst itself has no proper inner lining. In the lumen of the cyst there are some

CASE NO. 3.

Note the typical appearance of the dermoid representing the cornea. The lens is displaced and tilted. There is no communication between the interior of the globe and the degenerated plug of tissue representing the optic nerve. Retinal elements are gathered behind the posterior lens capsule.

well developed sebaceous glands. Their acini are full of some faintly staining structureless material, probably epithelial debris and secretion. The lumen of the cyst is divided into compartments by fibrous tissue septa. The rest of the cavity is full of loose areolar tissue which contained fat in the living specimen. The cystic wall

interposed between it and the optic cavity may be regarded as representing the cornea. As already stated, it resembles sclerotic in structure, though only one-third its thickness. At the middle of the posterior surface of this altered cornea is a thick line of homogeneous structure, probably representing Descemet's membrane, but there is no definite posterior endothelium. At the extreme periphery on the right side the only remnants of the iris are attached to the posterior surface of the cornea. This is the pigmented epithelial line whose posterior border merges insensibly into the choroid. Its anterior border extends only a short distance past the limbus corneae. Of course, there is no anterior chamber or angle, and the spaces of Fontana, etc., are also absent. The writer could not see any trace of the venous channel (Schlemm's canal) in any section. The space between the posterior surface of the cornea and the capsule of the lens is reduced to a mere chink. The lens is on the small side in all the slides, and is tilted so that its right equatorial extremity reaches further forward than does the left. This point is not well shown in the figure, as the lens is more oval than that which the artist has drawn. It is enveloped by a capsule which is closely applied to it posteriorly, and to its right side, but on reaching its anterior surface there is an interval between it and the lens which increases as we pass on to the left hand side of the slides. Here the capsule is actually folded up. Between the capsule and the lens in this situation are found embryonic fibres of the lens in different stages of development. Some cells are degenerated and vacuolated. Retinal epithelium is adherent to the posterior surface of the lens capsule. The sclerotic is well developed and appears quite normal, save for the pigment that is scattered all through its thickness. It is prolonged as a cord posteriorly. The outer covering of this cord is scleral tissue which is lined on the inner surface by a layer of choroid intermixed with retinal epithelium. In this layer are found some blood-vessels. Internal to this is a space which resembles the vaginal space of the optic nerve. Inside this is a plug of homogeneous matter, due probably, to the hyaline degeneration of embryonic mesoderm and nervous structure; it stains evenly and shows no structural details. Its surface is full of haemorrhages. This plug or cord of structureless material passes inside the cavity which represents the vitreous space, and spreads out to fill the posterior quarter of that cavity. Its anterior end contains numerous haemorrhages and is lined by the choroidal pigment. This fact is very well shown in the diagram, and in none of the slides could I make out any communication between the interior of the globe, *i.e.*, the part lined by the choroid, and this plug of degenerated fibrous tissue. Choroid lines the sclerotic and consists of an irregular network of pigment epithelium, fibrous tissue, and blood-

vessels of all sizes, cut across in different directions. There is a slight attempt at the formation of a ciliary body on the right hand side in the slides. Here the depth of the choroidal structure is increased and from its anterior part is given off the thin line representing the iris which is adherent to the posterior surface of the cornea. Rétina and remnants of the vitreous body are in the form of a solid mass filling the cavity of the globe, and immediately behind the posterior part of the lens capsule the embryonic retinal epithelium has the appearance of numerous tufts cut across in various directions; each of these has a central cavity filled by colloid matter that stains evenly and has no structural details. There is no suggestion of any pigmentation in this part of the slide. Posterior to the lens and about the middle of the cavity of the globe is a piece of hyaline cartilage cut across. It shows the usual structure, and is surrounded by embryonic retina. The rest of the cavity is full of embryonic undifferentiated retinal epithelium with haemorrhages in several parts and structureless colloid matter. In parts there are scattered amongst the retinal epithelium structures resembling malformed fibrous tissue. This seems to be prolonged forwards between the lens and ciliary process on the right hand side in the slides, and it appears that the dislocation of the lens in the opposite direction, is due to this.

Let us study each of these abnormalities separately.

Corneal Dermoid

Mitvalsky[10] who studied this subject very carefully gives the following figures. Out of 75 cases on record 90 per cent. were typical, that is, they were not anatomically connected with the lids, and in the majority of these the growth was situated at the corneo-scleral junction. In 5 out of these 65 the tumour was entirely corneal and in 10 entirely scleral. In some, conjunctiva was replaced by tumour tissue, in others it covered the growth, but in most instances a portion of the growth was apparently covered by conjunctiva, while the rest exhibited on the surface a pavement epithelium, papillae, hair follicles, and fine hair; sebaceous glands were common, but sudoriferous glands rare. According to the above writer the cystic form or closed dermoid is never met with in the eye.

The specimen above described is, of course, a typical dermoid. It is mainly corneal though it encroaches on the limbus. There is no trace of any normal corneal epithelium, but instead it is covered by a pavement epithelium, papillae, hair follicles and well formed hair. Sebaceous glands are well formed and the lumen of the cyst is full of fat and loose areolar tissue. On going through

the literature on the subject the writer is unable to find another case that showed all these points so distinctly and where the dermoid tumour was of such dimensions.

The tilting of the lens requires special mention. In the present case I cannot find any distinct cause for this displacement of the lens, save the fact that the embryonic retinal tissue seems to be adherent to the capsule on its posterior part, especially on the left side; then the embryonic fibrous tissue seems to be adherent to this part of the retina, and is continued on towards the back of the cornea between the lens and remnants of the ciliary body on the opposite side, which may possibly explain the present phenomenon.

Mayou[11] described a case in a child who was born at the seventh month. There was atypical fibrous development in the vitreous space, which had caused a backward dislocation of the malformed lens, with under-developed ciliary processes.

Treacher Collins[12] described a case in a microphthalmic eye where the lens was held back by the fibrous tissue in the centre of the vitreous body; the suspensory ligament of the lens was stretched and attached to elongated ciliary processes. Retina in this case was much folded.

Carl Hess[13] described a case of a microphthalmic eye in a child, aged three years. The measurements of the globe were 10 millimetres in all diameters; sclerotic, choroid, and retina were normal. There was a small coloboma of the iris. A process almost as large as the optic nerve came forwards in the vitreous cavity. This consisted of hyaloid artery with loose connective tissue containing mostly spherical nuclei and a few spindle-shaped elements. It decreased in size towards the centre of the vitreous to increase again further forwards becoming more fibrous in character as it reached the lens. It spread over the posterior capsule as a thick vascular membrane and passed as a delicate band round the equator of the lens through a coloboma of the iris into the anterior chamber. Here it split into two portions, one of which spread over the anterior capsule and was directly connected with the iris, and the other turned downwards to unite with sclerotic and choroid in the position of Fontana's spaces. The ciliary body, otherwise normal, was pushed away from the lens and bent towards the vitreous, at the place where this band came in relation to it, and the lens itself was dislocated in the contrary direction. No signs of any inflammatory processes were present. The cases quoted above would suggest that the dislocations of the lens that are present in microphthalmic eyes are usually due to some defect in the development of the vitreous body.

The piece of cartilage.—This piece of cartilage as described above is surrounded by the retinal elements, and on dissecting half the eye a note was made that this cartilaginous nodule is pushing the retinal elements from behind so that it has got inside the retina which is folded all round it. There are three possible sources for the development of cartilage in microphthalmic eyes.

(1) Most commonly it is due to an atypical development of the embryonic mesoblast that goes to form the vitreous body.

(2) It may be an atypical development of the mesoderm that forms the choroid and sclerotic and may push the retinal elements from behind, which get folded over it, and thus in a section the cartilage is surrounded by embryonic structure. This is what I think has occurred in the present case.

(3) It may develop in a fibrosed part of the retina, but this implies first of all intrauterine endophthalmitis, and fully formed blood-vessels; a condition not proved to occur. This question of cartilage in the vitreous cavity will be further discussed in connection with the next case which has two cartilaginous nodules present.

Undifferentiated embryonic retinal elements.—As stated in connection with Case No. 2, for the proper development of retinal elements there must be enough space for them to spread. In microphthalmic eyes on account of the atypical development of the embryonic mesoderm that goes to form the vitreous body, there is very little space for the retina to spread out, with the result that it gets folded and forms tufts that are mostly situated near the posterior surface of the posterior lens capsule. The next specimen to be described exhibits this deformity in a still more advanced form, and I will discuss the further facts in connection with it. On account of the absence of the ganglion cells of the retina and their axis cylinders, the optic nerve fibres, the hypoplasia of the optic nerve is easy to understand. In the present specimen the optic nerve and sheath are represented by the plug of colloid matter well shown in the figure and described above. It is impossible to make out any structure in it save some haemorrhages. It may be that it is degenerated glial elements or again it may represent the fibrous cord which originally held the arteria centralis. The axis cylinders of the ganglion cells of the retina are really neurons of the second order so that the optic nerve is not like any other cranial nerve. The neurons of the first order are the elements uniting the rod and cone layer to the ganglion cells. Its development proceeds from the cavity of the globe towards the brain, *i.e.*, the axis cylinders of the ganglion cells of the retina as they grow are gathered together and leave the eye as the optic nerve. As regards the development of their myelin sheath, this is derived from the brain (Mayou[14]).

Case No. 4. Second Eye from the same animal as Case No. 3

The eye is distinctly smaller than the previous specimen. There is an obviously small cornea, which is quite flat instead of being

CASE NO. 4.

Note absence of lens and pupillary opening. The anterior chamber is a mere slit. The coloboma of the ocular membrane is shown in the lower part of the figure (left side).

convex anteriorly. In circumference it is greatly reduced, being one-eighth of the circumference of the globe. The eye was cut into two halves as usual; one half was kept as a museum specimen; of the other half sagittal sections were cut serially.

The museum specimen shows defective sclerotic posteriorly. There is no sign of optic nerve present. On one side, which I

take to be the infero-medial, the sclerotic is represented by a very thin lining of fibro-muscular tissue. It is bulged outward by a plug of the contents of the globe that is protruding through the defect in the choroid and sclerotic at that place. There is a small

CASE NO. 4.

The cornea is shown on the left side in the diagram. Note coloboma of choroid and ectasia of sclerotic in the lower part of the diagram. There is a piece of cartilage in the vitreous cavity.

plug resembling the contents of the globe that lies outside the cavity altogether. The globe itself is very irregular in shape. Inside the cavity of the globe one can see a piece of hyaline cartilage. There is no trace of any lens discoverable. There is an obvious gap in the choroid on what I take to be the infero-medial surface. On the opposite side to this gap there are two

pockets formed in the sclerotic and choroid which contain structures resembling the folded up retina, like that found in the cavity of the globe. The retina is folded up and gathered into a mass that lies in the middle of the cavity. During the dissection of the half of the globe a note was made that a plug of fibrous tissue is uniting the ectatic sclerotic to the piece of cartilage that has been described above as lying nearly in the middle of the globe.

The thickness of the sclerotic varies. It is at its maximum at the limbus and a short distance away from it; as has already been stated, it is very defective posteriorly.

Microscopic description.—The slides to be described are stained with haematoxylin and eosin:

Cornea.—This is comparatively small in circumference, and, as already stated, is flat instead of being convex anteriorly. The corneal epithelium appears to be quite normal; it is seven cells thick. The cells are of pavement type anteriorly, but as one traces them further back they get more and more elongated, until we get to the line of cells next to the substantia propria, which are cylindrical.

There is no Bowman's membrane present. Substantia propria consists of the usual structure, that is, bundles of corneal lamellae arranged in a regular manner; the spaces between these lamellae are well marked. Posteriorly about the middle of the cornea Descemet's membrane can be made out. This seems to be lined by the posterior endothelium. There is an abrupt change in the structure of the corneal lamellae as we reach the limbus. The anterior chamber is a mere slit-like opening between the imperforate iris and posterior surface of the cornea. On one side of the slide the iris is adherent to the back of the cornea. The normal structure of the angle of the anterior chamber is wanting, that is to say, there are no spaces of Fontana, etc. The iris on the right hand side of the slide is composed of two distinct parts. The part adjacent to the remnants of the ciliary body is of great thickness and has a well-developed posterior pigmented epithelial layer (pars iridica retinae). Anteriorly the pigmented stroma cells are condensed to form another pigmented layer, which on a superficial examination resembles the posterior one, but under a high power has proved to be just a condensation of the pigmented stroma cells. Between the two layers above described are numerous blood-vessels of different sizes cut across in different directions. They have fairly thick walls and some of these contain blood corpuscles in their lumen. The stroma itself is composed of loose fibrous tissue and of chromatophores. The part further away from the ciliary body is very thin as compared with the part above described. It is composed of the same two layers, anterior

and posterior, of pigmented cells. Between these two layers in all slides there is a mass of red blood corpuscles that have gone on to hyaline degeneration, but the structure can still be made out. This part of the iris is adherent to its fellow on the opposite side. The iris on the other side is very rudimentary. As stated above, one part of it is adhering to the back of the cornea, while the other part dips backwards in between the embryonic retinal tissue. The ciliary body is represented on both sides by an irregular network of finger-like processes which are lined on the inner surface by pigment epithelium and contain in their lumen various amounts of fibrous tissue and colloid material. The choroid is very irregular in thickness in different parts of the slides. It is defective on the infero-medial side, *i.e.*, there is a coloboma of the choroid at this spot. Its normal structure is best seen where it lines the two diverticula situated opposite the colobomatous area. Here its innermost layers are composed of pigmented cells arranged in a regular manner resembling palisade epithelium, but the cells in the present case are more rounded. Exactly opposite the colobomatous area the choroid dips into the cavity of the globe in a long finger-like process; its end is directed towards the colobomatous area. From the slides it appears as if the fibrous tissue that is uniting the ectatic sclerotic with the cartilaginous pieces is prolonged forward to be adherent to the extremity of this choroidal incursion, and that it is dragging the choroid towards the colobomatous area at this spot. In structure the choroid is composed of blood-vessels of different sizes: some of these contain blood cells in the lumen, chromatophore cells and fibrous tissue. There is no lamina suprachoroidea discoverable.

The retina and the vitreous body.—As already stated the retina is folded up in a mass in the centre of the cavity of the globe and is very abundant just behind the iris to which it is adherent. In structure it is composed of embryonic retinal epithelial cells that line the sides of the finger-like processes, the lumen of which is full of colloid matter. These are cut across in various directions and give a very complicated picture. In the middle of the slides the retinal epithelium gives an appearance like that of the rosettes of a glioma. There are concentric layers of epithelial cells arranged round a cavity which contains blood cells. The vitreous body is represented by ill-developed embryonic fibrous tissue, and opposite the coloboma of the choroid are two nodules of hyaline cartilage. These cartilaginous nodules are united to the ectatic sclerotic by fibrous tissue containing retinal epithelium in places. In the cavity of the globe are found some haemorrhages. The two pockets described above are lined by the choroid, as already stated, and contain in their lumen huge masses of red blood cells with a few polymorphs.

The coloboma of the ocular floor.—Its size is different in various slides. If we trace it from the periphery towards the centre, in some slides it is not present at all; then in the innermost slides it is nearly one-seventh of the circumference of the globe. The ectatic sclerotic opposite the colobomatous area is very thin indeed. All these details are very well shown in the accompanying figure.

The important point to note is the fibrous tissue cord that seems to be uniting the cartilaginous pieces to the defective sclerotic and is prolonged forwards to be attached to the finger-like incursion of the sclero-choroidal tissue which it is dragging towards the coloboma.

There is no trace of any lens discoverable in any of the slides.

In this microphthalmic eye there are the following interesting points for discussion:

Absence of the lens.—This may be either due to absorption of the foetal lens or to its non-formation. In the case described by Hermann Becker and quoted above, there was no lens discoverable. Although Becker admits that the foetal lens may disappear completely, he believes that no lens was formed in his case, because if such had ever existed a pupillary opening must have been produced, unless of course the lens had developed in some other position and of this he could find no evidence. Mayou[14] describes a case of a child born at the seventh month who lived for twenty-four hours. He says: "In the right eye the condition represents what I take to be the failure in the secondary optic vesicle or lens to hit off the primary outgrowth from the fore-brain, with the result that the lens lies below the primary optic vesicle, the two layers of which have not come together since they have become distended to form a cyst, the anterior end of which lies in front and to the inner side of the lens. It is clear from this case that lens sometimes develops in other positions than the interior of the optic cup."

In the present case I believe there was no lens formed at all, otherwise one would expect to find a pupillary opening and some trace of an anterior chamber.

Coloboma of the membrane of the optic floor with ectasia of the sclera in that situation.—These colobomata are undoubtedly due to the defective closure of the choroidal fissure (foetal cleft). This cleft, as already stated, is formed simultaneously with the formation of the secondary optic vesicle and invagination of the lens. It is ventral in position and extends back a short distance in the pedicle. The cleft soon closes, but there is delayed retinal pigmentation at this spot (sixth week in man). When the eyes are gradually pushed from the lateral to the medial position, there is no rotation, so that the choroidal fissure still occupies the ventral position (Chievitz[15]). It is extremely rare to meet with an atypical

choroidal coloboma, though it does occur, and two choroidal colobomata may occur in the same eye. This is explained by the occasional occurrence of a double foetal cleft. A case of this kind was described in a calf embryo by van Duyse. As to the causes of the defective closure of the foetal fissure there are three different theories, *i.e.*,

(1) Mesoblastic theory.

(2) The retinal theory.

(3) The lenticular theory.

The lenticular theory which ascribes the defect in the closure of the foetal fissure to the abnormally big size of the lens, which fills up the cavity of the globe cannot operate in the present case on account of the absence of the lens. The second theory that ascribes the whole phenomenon to an undue proliferation of the retinal elements does not explain all the facts, and so I am leaving that out of discussion. In my mind the mesoblastic theory, as stated, explains all the facts very satisfactorily. v. Hippel's[16] experiments (quoted by Sir John Parsons) are very interesting in this connection. According to this writer the edges of the foetal cleft are separated by a narrow band of vascular mesoderm which increases in size. The cells in the lips of the cup are absolutely normal. As the development proceeds a struggle between these two tissues ensues; if the vitreous is normal then the retina spreads out without folding; if it is diminished the retina is folded and endeavours to find space by overlapping the edges. If the vitreous is very small, folding of the retina becomes much pronounced. As explained while discussing Case No. 3 the amount of vitreous depends on the intruded mesoderm; if it is dense very little vitreous is formed and *vice versa*. This theory seems to explain all the facts found in colobomata of the choroid and retina.

Cartilaginous nodules which are present.—It is clearly seen from the diagram and slides that the cartilaginous nodules are arising as atypical development of the intruded mesoderm that goes to form the vitreous body. In this connection work done by Carl Hess is very important. One of his cases where the lens was pushed on one side has been quoted above while discussing Case No. 3. That showed an atypical development of vitreous with choroido-retinal and iridic colobomata. There was no sign of any inflammatory process present. Case No. 2 in his series represented bilateral microphthalmia. The lenses in both eyes were fixed against the ocular tunics in close proximity to the optic nerve. There were colobomata present as in the other case. Connective tissue bands were present in the vitreous. The Case No. 3 was again a bilateral microphthalmia in a prematurely born child. Vitreous was pierced by a band of fibrous tissue surrounding the

hyaloid artery forming the capsule of a dislocated irregularly shaped and cataractous lens and proceeding forwards in the anterior chamber to join the corneo-scleral border. Its anterior portion contained hyaline cartilage. The right eye contained a similar piece of cartilage. Other cases of atypical development of the vitreous have been quoted above (Mayou, etc.).

Let us take the last interesting feature of this case, *i.e.*, the undifferentiated embryonic retinal tissue portions. As stated above the mal-development of the retina is the result of defective vitreous cavity. The appearance of rosette formation is really due to folds of retinal elements having been cut across in different directions. It is quite a common occurrence in microphthalmic eyes. Cases have been described by Greeves[16] and others.

In the second case described by Greeves in a patient, aged 4½ years, the right eye had been much smaller than the left since birth. There was no history of any eye trouble. The retina was gathered into a mass in the centre of the globe. In some parts an appearance of rosettes was produced by a section across the convolutions. Lens in this case was represented by a shrunken mass of capsule lying in the centre of the pupillary area.

Summary

I have described above four cases of microphthalmia representing different stages at which the arrest of normal development has taken place. In all these cases aberrant development was present. There are two main theories to explain this abnormality. Deutschmann[17] attributes all congenital abnormalities to intrauterine inflammation. This may operate in cases of atypical colobomata of the iris and of anterior staphylomata, but it has never been proved to have occurred in cases of microphthalmia. There is no suggestion of any inflammation in the cases described above. Hess describing four cases of microphthalmia, to which reference has already been made, came to the same conclusion. In none of his series of cases was there a suggestion of any inflammation.

The second theory attributes this abnormality to an arrest of normal development. Any deleterious agent might be expected to act injuriously on delicate foetal structures. In this connection Ochi's[20] experiments are interesting. He employed four methods :

(1) Salt solution injected near the blastoderm.

(2) Distilled water injected near the blastoderm.

(3) Air injected near the blastoderm.

(4) Mechanical disturbance.

By these means he obtained microphthalmia in 26 cases.

There is strong evidence that in Case No. 1 of our series fright has been the operative cause. First cessation of the menses usually corresponds to about three weeks' gestation and this is the time when the optic vesicles are being formed as a pair of hollow diverticula from the lateral aspects of the fore-brain. This coincides with the time when the girl first realized her condition, and the consequences associated with it. Naturally, her whole nervous system is upset with this sudden shock, and it is quite conceivable that this acts deleteriously on the developing embryo with its maximum effect on the most delicate structure, that is, the primary optic vesicle. It may be that no primary optic vesicle is formed at all or that its future development is arrested. Probably in other cases some such agent as an injury, change in pressure, chemical poison, etc., has been operative.

Consanguinity is sometimes blamed as the cause of these abnormalities. Cases of anophthalmia have been described by H. Triepel[18] and Cecchelto[19], and microphthalmia by van Duyse, but of course, this is not always found.

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CONGENITAL MUSCULAR SQUINT

BY

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THERE is a peculiar kind of squint, which is not concomitant, and has the following characteristic signs:

The movements of abduction of one or both eyes are either abolished or greatly reduced. *The movements of adduction* may be impaired or they may be normal; but they are always associated with a retraction of the eyeball into the orbit, and a closing of the palpebral fissure. The eye retracts more or less strongly into the orbit, and the palpebral fissure becomes narrower as the adduction of the eye increases.

This variety of squint is always congenital, as the acquired type, due to adhesions in the orbit, caused by cysts, inflammations, etc., and by cerebral disease, can scarcely be classified in this group.

A very complete study of this condition has been made by the late Professor Lagleyze of Buenos Aires, in his book *Du Strabisme*, published in Paris in 1913. He described five cases, two of which were limited to one eye, and the other three to both eyes. Other observers have studied this condition later, and Bietti was able to collect 31 cases published in ophthalmic literature. Duane published a record of 38 cases, of which 14 were his own. Since Lagleyze's time I have only seen one case, and Adrogué has collected seven cases seen at the Ophthalmic Hospital in Buenos Aires; some of his cases seem to be very doubtful and would be better diagnosed as absence of or congenital paralysis of the superior oblique, quite a different entity from the one under consideration.

In October, 1923, I was consulted by Robert S., an Argentine student, aged 16 years.

Examination.—Left eye convergent strabismus of 15°.

When made to look to the right, a deep retraction of the left eyeball into the orbit took place, and the palpebral fissure closed. The farther he looked to the right, the greater was the retraction and the narrower the palpebral fissure. When made to look to *the left,* the eye remained exactly in the middle line.

Diplopia did not exist in the primary position; but became manifest when the eyes were turned to the left.

Enophthalmos of a very slight degree existed in the left eye, whilst the palpebral fissure was narrower than that of the right eye. The measurements of the fissures were: R.E. 8 mm.; L.E. 6 mm.

In forced adduction the fissure was narrowed to 3 mm. and there was a sinking of the eyeball into the orbit of about 5 mm.

In abduction the eye appeared to advance slightly.

The active movements of the left eye were: out 0°; in 10°; up 40°; down 40°.

The passive movements obtained by the use of fixation forceps were: out 5°; in 10°; up 40°; down 40°.

The movements of the right eye, active and passive, were quite normal.

Refraction examined with atropin. R.E. +1.50 sph. +2.50 cyl. axis 90°; V.=6/9. L.E. +1.50 sph. +2.50 cyl axis 90°; V.= 6/12.

Diagnosis: *Congenital muscular squint of the left eye.*

Operation: Left eye, tenotomy internal rectus. The tendon and its insertion appeared to be normal; but on making traction upon it with the strabismus hook, it gave the impression of being a thick inextensible cord. The external rectus was not touched.

Result: The cosmetic effect of the operation was good and satisfied the patient. The retraction of the eyeball which existed in the primary position disappeared; the lateral movements of the eye were unchanged, and the retraction on forced adduction still persisted. The palpebral opening was widened and appeared to be more or less the size of the right one.

Pathogenesis

The exact cause of this variety of strabismus is still unknown, and its two characteristic symptoms must be studied separately.

The absence of the movements of abduction is not due to a paralysis of the external rectus, because even with a fixation forceps, the eye cannot be drawn out; nor is it due to the congenital absence of the muscle, because I have been able to detect muscular fibres in two of the cases that Lagleyze operated on and Adrogué has also noted their presence in cases operated upon by him.

The retraction of the eyeball into the orbit is not easily explained. Certain mammals have a retracting muscle which pulls the eye back; but it has never been found in man. Some observers, such as Axenfeld, consider the retraction to be due to an inextensible ligament, which is inserted behind to the orbital wall, and extends forward to the eyeball, fixing it in a certain position and impeding the action of the muscles, although they may be quite normal. Lagleyze searched very carefully for this ligament and was not able to find it in any of the cases operated upon by him.

Other observers consider it to be due to a very short and powerful internal rectus, inserted near the posterior pole of the eye; but I have not found this to be so, as in the case I operated upon, though the tendon was very stout, it was inserted in its usual place.

Histological changes exist in the muscles and in the aponeuroses. There must be an abnormal formation of fibrous tissue in the

muscles and a thickening of the aponeuroses, which either abolishes or greatly diminishes muscular contractility. Most of the symptoms observed in this variety of squint, could be explained by the formation of fibrous tissue. The diminished lateral movements of the eye, by the absence of muscular contractility; and the retraction of the eyeball, by the shrinking inherent to all fibrous tissue, and consequently to the shortening of the internal rectus, and the loss of elasticity of the external rectus.

The literature on the pathology of this disease is abundant; but the conclusions of Alling, Green, Türk and Bahr are unsatisfactory.

The condition of the sclerotic in all cases of congenital strabismus is interesting. Schoeller remarked that it was extremely thin; and in eight cases operated upon by him, he had three cases of perforation during tenotomy or advancement; and this was in no way attributable to faulty technique as in over 2,000 operations for ordinary squint, this accident never occurred. Both Axenfeld and Lagleyze have had similar accidents. In the latter's case he was demonstrating to the students the great resistance offered by the internal rectus, by making traction upon it with a strabismus hook, when he was disagreeably surprised to find that the sclerotic had given way just beneath the insertion of the muscle. On examination of the sclerotic it was found to be very thin and of a pale blue colour.

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ANNOTATION

Institutional Treatment of Eye Disease in Children

In the treatment of interstitial keratitis the benefit of any special measures may be open to discussion. There can, however, be no doubt as to the necessity for seeing that the patient is placed under the best hygienic conditions. Even if these were obtainable in our Metropolitan Hospitals the pressure on the available accommodation would prevent their use for such a chronic complaint.

Yet the difference between adequate and inadequate treatment in this disease may mean the difference between a useful and a useless citizen. In 1924, owing to the diminution in contagious eye diseases in the Metropolitan school district, it was found practicable to devote some space to cases of interstitial keratitis at White Oak, Swanley. Mr. Treacher Collins, Consulting Surgeon to the Hospital since its start as a trachoma school in 1903, states in the Children's Section of the Metropolitan Asylums' Board Annual Report for 1925-1926, that: "the result has proved eminently satisfactory. Twenty-four new cases were admitted and nineteen were discharged. In these latter the inflammation had entirely subsided without, in any case, leading to the incurable blindness which frequently ensues when treatment is neglected or inefficiently applied." He further adds that if the exceptional advantages which White Oak now offers for dealing with this distressing class of case were more generally known many more cases would be sent there for treatment. We trust that our readers in the Metropolitan area will bear this recommendation in mind.

The institutional treatment of trachoma and phlyctenular conjunctivitis in children has proved so eminently successful that its extension to cases of interstitial keratitis is to be heartily welcomed. The opportunities at present afforded are meagre considering the number of cases, especially as regards phlyctenular disease. Nothing succeeds like success and we confidently hope that even greater facilities may be afforded in the future.

ABSTRACTS

I.—ANATOMY AND PHYSIOLOGY

(1) **Dejean, Ch.—Anatomical and embryological study on the mammalian hyaloid; its rôle in the formation of the ocular media, its limits and structure. (Étude anatomique et embryologique sur la membrane hyaloïde de l'oeil des mammifères. Son rôle dans la formation des milieux de l'oeil, ses limites, sa structure.)** *Arch. d'Ophtal.*, Vol. XLIII, No. 5, May, 1926.

(1) In this paper, which is a continuation of **Dejean's** previous work on the origin of the vitreous, the question of the staining reactions of the various parts of the developing eye is discussed. An attempt is made to settle the perennial arguments as to the mesodermal or ectodermal nature of the vitreous and the presence or absence of a hyaloid membrane. The paper is divided into sections as follows:

(1) Proofs of the existence of the hyaloid membrane. Macroscopic investigations on fresh eyes in various mammals are described, and also microscopic appearances, stress being laid on the necessity for perfectly tangential sections.

(2) Origin and development of the hyaloid membrane. This is the most important section of the paper. In it Dejean describes his findings in early embryos of rabbit, sheep, and other vertebrates. He employed differential stains designed to show the presence or absence of collagen, and he takes the presence of the particular colouration involved to indicate the mesodermal origin of the tissue so stained. In all cases he found a surface membrane on the developing inner layer of the optic cup which gave a collagen reaction. This membrane was continuous with a similar one on the surface of the lens vesicle and as lens and retina separated these membranes widened, became less dense and formed the primitive vitreous. This he therefore considers to be mesodermal in origin. He states that he was able to distinguish, in older embryos, between remains of this membrane—the hyaloid proper—and the internal limiting membrane of the retina. He traces the hyaloid membrane anteriorly on to the ciliary body and observes the development of the zonule fibres from it in this region. As development proceeds a secondary vitreous is formed from the membrane in the region of the retina, which displaces the primitive vascular vitreous towards the lens and towards the centre of the eye. The line of contact between primitive and secondary vitreous undergoes condensation and forms an "intervitreous" limiting membrane which lines the patellar fossa and the canal of Cloquet, and separates the vitreous proper from the zonule region. The anatomical arrangements are described in detail, and three membranes are recognized:

(*a*) The hyaloid membrane proper, which lines the retina as far forwards as the ora serrata, to which it is strongly adherent.

(*b*) The limiting membrane of the ciliary region, which is the anterior portion of the hyaloid proper, and covers the ciliary processes and the anterior surface of the zonule.

(*c*) The intervitreous membrane which runs from the ora serrata to the lens and turns backwards to line the canal of Cloquet.

(3) The anatomical peculiarities of the adult hyaloid and the consequences from the point of view of operation. This section deals with the points of attachment and the general disposition of these membranes in the adult and discusses the importance of the intervitreous membrane in intracapsular extraction.

(4) A short section on the structure of the hyaloid membrane. This deals with the adhesion of the vitreous fibrils to the membrane and its condensed nature.

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(2) **Dejean, C. (Paris).—On the formation of the structure of the vitreous in the eye of vertebrates. (Sur la formation des milieux figurés de l'oeil des vertébrés.)** *Arch. d'Ophtal.*, Vol. XLI, p. 659, 1924.

(2) In this paper **Dejean** deals with the much disputed question of the origin of the vitreous. He summarizes the views of previous observers, dealing in the first place with the older view that the surface ectoderm of the head is originally separated from the optic outgrowth by mesoderm, which therefore becomes compressed between the two as they approximate. He shows that (as is now accepted by most anatomists) in reality the optic outgrowth can be recognized as a pit on the floor of the wide open neural plate while this is still directly continuous with the surface ectoderm, and that at this stage the two ectoderms are in direct contact, no mesoderm intervening. He considers the alternative views that :

(1) There is an intermediate layer of connective tissue separating lens plate from optic vesicle and that this layer forms the vitreous (Kölliker).

(2) Into a mesodermal separating layer grow ectodermal fibrils from the lens vesicle and the retina (van Pee), and that

(3) the vitreous is entirely ectodermal and is derived from filaments which directly connect lens and retina (Laguesse, v. Szili, Mawas and Magitot).

He has himself studied embryos of mammals, birds, reptiles, and fish. He considers that the small isolated cells found between the lens and retina in early stages are entirely vaso-formative in nature. They occur in much greater number in animals which have a large and well-developed hyaloid system. The more or less homogeneous medium in which they are embedded gives the staining reactions of collagen very strongly at a time when the rest of the mesodermal connecting substance is in the stage of pre-collagen. He considers that the appearance of protoplasmic bridges between lens and retina is chiefly an artefact due to shrinkage and vacuolation of the cells and that the primitive vitreous is developed from the thickened basement membranes of lens vesicle and retina. He considers that a secondary vitreous body develops later in mammals, from the retinal basement membrane only, and displaces the primitive vitreous towards Cloquet's canal and the back of the lens, and towards the ciliary region (where it forms the zonule).

He ends by stating that chemical researches on the adult vitreous show it to resemble chemically other connective tissue substances, but to contain a higher percentage of mucin. It does not show the reactions of neuroglia, nor does it contain elastin or gelatin.

It presents the characters of embryonic connective tissue. The vitreous is therefore a true connective tissue substance specialized for a definite function, by arrest at an embryonic stage.

IDA C. MANN.

(3) **Halbertsma, K. T. A. (Leyden).—Two cases of microphthalmos with orbital cyst. (Deux cas de microphtalmos avec kyste orbitaire.)** *Arch. d'Ophtal.*, November, 1925.

(3) **Halbertsma** in this paper has had two specimens of microphthalmos of which he has made an exhaustive study. In his first case the eye was not removed until the patient was aged 19 years; the measurements of the globe were $12 \times 10 \times 10$ mm., and those of the attached cyst $36 \times 20 \times 10$ mm. The second specimen was obtained from a child, aged 9 weeks. In each case the fellow eye was normal. The cases appear to be fairly typical and do not present any unusual features. The anatomical and histological characters are described in full detail and form interesting reading. The author has evidently expended much time and care in the preparation of microscopic sections, but the reproduction of his microphotographs are so poor that few of them are of much help to the reader. Halbertsma discusses the pathogenesis of this developmental anomaly at considerable length but his views cannot be adequately represented in an abstract. Those interested will do well to consult the original.

J. B. LAWFORD.

(4) **Graves, Basil (Liverpool).—Change of tension on the lens capsules during accommodation and under the influence of various drugs.** *Brit. Med. Jl.*, January 9, 1926.

(4) In this paper, which was first read before the American Ophthalmological Society, **Graves** describes the slit-lamp findings in a very interesting case in which the entire lens substance had undergone absorption, leaving the lens capsule intact save for a minute hole, and with clear media. The original injury was probably due to a foreign body which was still to be seen as a tiny brown spherical object at the bottom of the anterior chamber. The eye with a +14D. sph. had a visual acuity of 6/5. Graves made numerous observations on the effects of accommodation and drugs on the capsule. The posterior part altered more in the nature of relaxation than the anterior part. He sums up as follows: "The behaviour of these capsules shows that one mechanical result in the process of accommodation consists in a relaxation of their state of tension. Further, if a mydriatic is given the capsules are rendered taut; and if time is allowed for these capsules to get just so taut that they do not relax when the patient gazes close, but not so taut that they fail to relax when the

other eye is pressed, and the fundus is at this stage flooded with a sufficiently strong light-stimulus to cause good contraction of the already moderately dilated pupil, the pupil-contraction thus produced takes place without causing any evident relaxation of the existing capsular tension, showing that tension of these capsules caused by the mydriatic is independent of the dilatation of the pupil which is also caused by the mydriatic. If, when the pupil is well dilated and the capsules are tense under the full influence of a mydriatic and cycloplegic such as cocain or homatropin, eserin is then instilled, in a very short time the capsules begin to relax and they then rapidly become extremely lax—far more lax than they ever were without the influence of a drug such as eserin—this occuring before contraction begins in the pupil which is still fully dilated by the mydriatic. When a stronger drug such as atropin is used, though longer time is required for the eserin action to begin to show itself by reduction of the capsular tension, even so this result occurs before the miotic begins to show its influence in diminishing the size of the dilated pupil. Tests made on other occasions which I have not quoted here confirmed that eserin acts on the capsular tension before it begins to act on the size of the pupil, when this eye has previously been subjected to the influence of a mydriatic."

E.E.H.

(5) **Ferree, C. E., Rand, G., and Wentworth, H. A. (Bryn Mawr, Pa.).—The blind spot for achromatic and chromatic stimuli.** *Amer. Jl. of Ophthal.*, August, 1925.

(5) **Ferree, Rand** and **Wentworth** open their paper with a survey of the literature on this subject. This shows a great deal of disagreement in the results obtained, in work on the size of the blind spot. The authors therefore investigated the subject with special apparatus and their usual meticulous care. They found (1) that the blind spot for achromatic stimuli averages 7.6 per cent. larger on a white than a black field, using a black target in the first instance and a white in the second. This phenomenon is due to irradiation produced by the white object or white background. (2) The size of the blind spot for chromatic stimuli is greater than for achromatic stimuli, and depends on the colour of the surrounding field: on a grey background of the same brightness as the colour, the enlargement was 40-45 per cent., on a black field 47-71 per cent., and on a white field 72-132 per cent. (3) The order of ranking in size is also affected by the background. From the largest to the smallest the order on a white field was green, blue, red, while for the grey and black fields, it was green, red, blue.

F. A. WILLIAMSON-NOBLE.

II.—MISCELLANEOUS

(1) **Sprague, White, and Kellog.—Disturbances of vision due to digitalis.** *Jl. of Amer. Med. Assoc.*, September 5, 1925.

(1) This is a report of seven cases which received an excessive dose of digitalis. In all but one case this was due to incorrect dosage by the physician in charge, or misunderstanding on the part of the patient. **Sprague, White,** and **Kellog** sum up as follows:

"Five patients complained of a defect of colour sensation; four had yellow vision, one red-yellow, and two green. All complained of decrease of visual acuity, and three said that they seemed to be looking through mist. Two had difficulty in focussing the eyes for reading, two had definite scotomas, and three had flickering before the eyes. In one instance the patient said that surrounding objects in sunlight appeared covered with snow.

"In two cases the visual disorders preceded the gastro-intestinal and cardiac effects; in two others, disturbances of the eye were associated with nausea and vomiting as the first toxic symptoms. Two patients with marked visual effects had no change in cardiac rate or rhythm, and one of these had striking xanthopsia without either nausea or cardiac effects.

"In three cases, extreme muscular weakness, described in digitalis intoxication, was a prominent feature. This, with the gastro-intestinal complaints, was considered by the patients of more serious importance than the eye symptoms; and it was therefore necessary to question them carefully to secure an adequate description of their visual disorders.

"Disturbances of vision resulting from the internal use of digitalis have been known at least from the time of Withering. Such disturbances have only rarely been described in the English literature, but have been carefully reported in German and French."

A. F. MACCALLAN.

(2) **Mills, Lloyd (California).—Eyedness and handedness.** *Amer. Jl. of Ophthal.*, December, 1925.

(2) The dominance of the right or the left eye and greater facility of using the right or left hand are designated as "eyedness" and "handedness." **Lloyd Mills** has made an interesting investigation into these conditions and the following are some of his findings. One eye always posseses a greater sense of clarity or discrimination, even when both are alike in refraction. The right is usually the better. This forms part of a general bilateral asymmetry of function, so that in the right handed, the centre of

gravity of the whole body is to the right. This causes one to walk, swim, or run to the right, in the absence of guiding marks. This asymmetry is probably of great advantage to mankind since it simplifies neuro-muscular control and inhibition. Eyedness determines handedness, the line of vision of the eye, chosen as the fixing eye, regulating the matter of dominance of the corresponding side of the body which it guards and controls. Signs of left handedness can be found in 20 per cent. of the population, frequently with a familial history. The figure indicates that sinistrality persists as a Mendelian recessive in spite of racial training to eradicate it. The characteristics of the non-master eye are, that it is cyclophoric, frequently hyperphoric, and that it diverges when a test object is brought closer than the convergence near-point. A useful object is the plane ophthalmoscopic mirror. This is held at a distance of two feet from the patient, who is asked to fix the hole in it. It is then steadily brought forward in the median line until one or other eye diverges. There are four groups of dextrals and sinistrals. (1) The pure dextral, right handed and right eyed, 76 per cent. of the population; (2) the pure sinistral, left handed and left eyed, 9.3 per cent.; (3) the crossed dextral—right handed (usually by training), and left eyed, 13 per cent.; (4) the crossed sinistral, left handed and right eyed, 1.7 per cent. The characteristics of these groups are modified early in life by injuries or diseases which greatly reduce visual acuity in the master eye. After the pathways establishing ocular dominance are fully developed, mastery may persist, however, in spite of great loss of vision in the master eye. Conical cornea usually appears first in the non-master eye, *i.e.*, in the eye which is on the weaker side of the body. Ocular mastery should be maintained with care in the correction of errors of refraction, particularly in myopia and anisometropia.

F. A. WILLIAMSON-NOBLE.

(3) **Adler, Francis, and Landis, E. M.—The effect of miotics and mydriatics.** *Arch. of Ophthal.*, May, 1925.

(3) This investigation was undertaken by **Adler** and **Landis** on account of the conflicting results obtained by Wessely and by Seidel. The former holds that the increase in protein content of the aqueous which follows instillation of miotics is due to increased permeability of the capillary walls, the latter that it is due to direct stimulation of the ciliary epithelium. In their earlier experiments the investigators followed Seidel in estimating the amount of protein by means of the refractometer. This method, however, was useless as it was susceptible of a hundred per cent. error, and by using the "nephelometer" method—described in the text—the experimental error was cut down to 2 per cent. Certain precautions

are necessary, *e.g.*, the aqueous must always be withdrawn in the same amount and at a uniform rate, and the iris must not be damaged. The findings, in which the figures give the average of several experiments on different animals, were as follows: (1) Two drops of 1 per cent. eserin instilled in one eye at half-hour intervals, and aqueous withdrawn from both eyes half an hour later. The eye which had had the eserin showed a 44 per cent. increase of protein in the aqueous; (2) a similar experiment using atropin showed no effect; (3) a similar experiment in which atropin was followed by eserin showed no increase in the protein content, *i.e.*, the effect of eserin was prevented by the previous use of atropin; (4) control experiment in which nothing was put in either eye, average difference 1.6 per cent.; (5) pilocarpin used, no increase of protein; (6) extirpation of one ciliary ganglion a month before—decrease of protein in the affected eye, 28 per cent.; (7) effect of eserin on the denervated eye—protein in aqueous, 11.4 per cent. above that in unaffected eye, *i.e.*, allowing for effect of extirpation of ganglion, total increase is $11.4+28.2=39.6$, a figure closely approximating to that found in experiment (1). The authors consider that these results are difficult to interpret, in view of the difference between the effect of removal of the ciliary ganglion and the instillation of atropin. They feel justified, however, in saying that eserin causes an increase in protein content by action on some other structure than the third nerve fibres running to the ciliary epithelium. Seidel's contention that the action of eserin is a proof of the secretory activity of the ciliary epithelium, is therefore not valid.

F. A. WILLIAMSON-NOBLE.

(4) **Cazalis, Jean, neveu (Beziers).—On the usefulness of miotics in the treatment of superficial keratitis, simple or ulcerative. (De l'utilité des myotiques dans le traitement des kératites superficielles simples ou ulcéreuses.)** *La Clin. Ophtal.*, May, 1925.

(4) **Cazalis** in the course of a short article advising a more general employment of miotics in corneal disease after atropin has been disappointing in its effects, utters a caution about the use of miotics which seems worthy of special notice. "In old people, and especially in our southern climates, even in the complete absence of hypertension and of any appreciable participation of the iris in the inflammatory process, miotics have sometimes a tendency to produce a kind of tetany (*tétanie*) of the iris with posterior synechiae; these adhesions to the anterior capsule are not without disadvantage and by the screen which they interpose between the anterior chamber and the posterior media they ultimately bring about the risk of glaucoma or of sympathetic

irritation." The author has never seen this complication in persons under 50 years of age.

ERNEST THOMSON.

(5) **Ramsay, A. Maitland (St. Andrews).—The symptomatology of disturbances of the visual function.** ***Glasgow Med. Jl.,*** November, 1925.

(5) There are various ways of discussing the diseases of an organ. The usual way is the textbook way of describing the diseases with their symptoms. Another way is to take the various symptoms exhibited and to correlate them with the diseases. In this lecture delivered at the opening of the winter session of the James Mackenzie Institute for Clinical Research, St. Andrews, **Ramsay** adopts the last-named method and one feels that a critical, though doubtless non-specialist audience, must have been greatly interested. Ramsay says: "It has always been my endeavour to bridge over the gap between narrow specialism and general medicine, because I believe that no progress can be made until it is recognized that the principles of general physiology are sufficiently broad and deep to include every organ of the body. To emphasize specialism is a retrograde step. It is always the same physiological and pathological processes that are at work." Ramsay's students all know how well he succeeded in this endeavour, and in this lecture, presumably to a general medical audience, there is a large amount of mixed wisdom, that of the physician and the specialist, and the specialist no less than the general physician may well be able to profit by it.

ERNEST THOMSON.

(6) **Ruata, Prof. Vittorio (Padua).—Nicotinism, nicotine amblyopia and the treatment of these conditions. (Considérations sur le nicotinisme, l'amblyopie nicotinique et leur traitement.)** *La Clin. Ophtal.*, January, 1925.

(6) To judge from **Ruata's** article tobacco poisoning must be a very serious trouble in the district in which he practises. Ruata says, in the first place, that tobacco-alcohol amblyopia ought to be called tobacco amblyopia because tobacco is really the determining agent. Alcohol is merely one of the aggravating agents, others being represented by overwork, poor diet, poor digestion, bad hygienic conditions and so on. In the second place, tobacco amblyopia should be considered as one of numerous symptoms of chronic tobacco poisoning, and as a symptom which is never primary but always preceded or accompanied by disorders of the cardio-vascular, digestive, and nervous systems. It represents an advanced and relatively serious stage of tobacco intoxication. Thirdly, the author seriously suggests that inveterate tobacco users

should be treated in the same way as morphinomaniacs and be interned in private hospitals (*maisons de santé*, which perhaps even means asylums) in which they should be treated under strict supervision not only by deprivation of tobacco but in other ways calculated to eliminate the poison and to improve the general health. With regard to the treatment of the general condition, Ruata, having tried all the usual drugs such as strychnine, lecithins, bromides, valerian, etc., has had great success with a preparation which he says is well known in Italy, being prepared at the Zoja laboratory, Milan. This preparation which was introduced in 1906 by Professors Vanzetti and Piccinini is a formate of tetramethyl-ammonium. It is a cardio-tonic of the caffeine type, a myo- and neuro-tonic of the strychnine type and also has a hypercrinic action, that is, it stimulates all the secretory functions.

ERNEST THOMSON.

(7) **Goadby, Sir K.—Latent sepsis**. *The Practitioner*, November, 1924.

Cotton, H. A. (New Jersey).—Effect of chronic sepsis upon the central nervous system. *The Practitioner*, May, 1925.

(7) The many pathological eye conditions resulting from latent sepsis are known to ophthalmic surgeons, but the method by which the poison acts is not so well recognized. **Goadby** explains this in his article. He mentions that digestion of bacterial protein either by autolysis or by tissue enzymes may result in poisonous products. Chronic infection of the tonsils and gums (pyorrhoea), old persistent infection of the mucous membrane of the uterus, the urethra, and above all, the lining membrane of the large bowel in chronic constipation and intestinal sepsis, appear to fulfil all the conditions necessary to promote bacterial autolysis and diffusion of the resulting poisonous compounds. These poisonous protein substances, originating from bacterial autolysis, are not specific poisons; they may be produced from the protein of any bacterial species, and from their special nature produce no immunity response, nor antitoxin. Sub-infection, where the bacteria gaining entrance are dealt with promptly by the phagocytes, must not be confused with latent infection, where the bacteria are out of reach of phagocytic activity. It is impossible to estimate the far-reaching effects of small but persistent foci of latent sepsis. It is not improbable—more, it is possible—that the insidious onset of arterio-sclerosis and the first breakdown of the kidney cells in chronic nephritis are but the sequels to absorption of unspecified bacterial poisons generated in sequestered and undetected places.

Cotton believes that there are two forms of toxaemia resulting from intestinal stasis. One type is the result of infectious processes

in the walls of the colon. The other form of intestinal stasis is due to generation of toxic products from the retained residue of the digestive processes. He is convinced that toxaemia is the cause of all the so-called functional psychoses, and he has successfully treated by detoxication 86 per cent. of the 1,600 cases which have been under his care. He believes that the principal factor is cerebral toxaemia, the result of chronic sepsis located in the teeth, tonsils, gastro-intestinal tract, and genito-urinary system. Proof is found in the fact that when such foci of chronic sepsis are eliminated early in the course of the disease, the mental symptoms disappear.

A. F. MACCALLAN.

(8) **Edeskuty, O.—Primary Necrosis of the Iris. (Ueber primäre Irisnekrose.)** *Zeitschr. f. Augenheilk.*, Vol. LV, p. 38, 1925.

(8) **Edeskuty** describes an interesting case of a shoemaker, aged 61 years, who, without any apparent cause, suddenly developed a severe inflammation in the left eye accompanied by a violent headache confined to the same side of the head and temple. After the first day vision completely disappeared from the eye. The cornea was dull, and the iris swollen and discoloured, and a considerable deposit of "k. p." was evident. The trouble progressed rapidly, and a fibro-purulent discharge filled the anterior chamber, leading to an increase of tension and much pain. An anterior sclerotomy was therefore performed, but this giving only temporary relief, the eye was finally enucleated.

Histological examination showed three areas of necrosis in the cornea, while the whole of the iris substance was in an advanced necrotic condition. Edeskuty considers that the microscopic findings were identical with those described by Meller (*Zeitschr. f. Augenheilk.*, Vols. XLIII and L) as occurring in cases of herpes, and such an explanation, in his opinion, would adequately account for the similar condition in the cornea: an atypical case of herpes with unusually severe ocular complications.

W. S. DUKE-ELDER.

(9) **Comberg, W., and Stoewer, E. (Berlin).—The mechanism of raised intra-ocular pressure on muscular contraction and its clinical importance. (Die Augendrucksteigerunde Wirkung verschiedener Muskelaktionen und ihre Bedeutung fur klinische Verhaltnisse.)** *Zeitschr. f. Augenheilk.*, Vol. LVIII, p. 93, 1925.

(9) In the German literature there are two views on the mechanism of the raised intraocular pressure which accompanies contraction of the orbital musculature: the one (Levinsohn, 1910), that the larger part of the reaction is due rather to incidental

causes such as vaso-motor disturbances, and that the action of the muscles themselves is without large practical importance; the other (Lederer and Wessely), that the orbital muscles play the preponderating part. **Comberg** and **Stoewer** claim to have substantiated the latter view in a series of experiments whereby the intraocular pressure, as taken by the Schiötz tonometer, was compared when the two influences were to some extent dissociated.

A series of comparative measurements of the intraocular pressure and general blood pressure (sphygmomanometer on arm) were taken on compression of the chest and thorax (Valsalva's experiment). Under these conditions the systolic/diastolic blood pressure rose from 90/120 to 120/160 mm. Hg, and the intraocular pressure from 18 to 30 mm. Hg (Schiötz); the whole of this rise of pressure is presumably circulatory in origin and it came on slowly and established itself gradually. Another series of experiments in which the intraocular pressure was taken on forcibly closing the lids showed a much higher rise—from 18 to 65 mm. Hg—and one which came on suddenly and disappeared immediately on stopping the blepharospasm; this effect is presumably largely due to the action of the muscles themselves.

Experiments were further done while the lids were thrown into voluntary spasm as in the act of closing while being held open by retractors. Here again a comparable rise of intraocular pressure was obtained, which the authors ascribe to the pressure of the pars orbicularis on the globe. No pattern of retractors could be found which were able to eliminate this effect, but it could be completely abolished (by paralysing the muscle) with an injection of novocain.

The clinical interest the authors attach to these experiments lies in the light they throw on the pressure conditions obtaining in an operation as for extraction of cataract. A series of experiments were done wherein the intraocular pressure was taken when the same amount of pressure was applied to the eye by a spoon as they conceived the average operator applies it to the cornea in the expulsion of the lens. The intraocular pressure rose from 18 to 30 mm. Hg. Since this occurred in the intact eye, the rise of pressure that takes place in an operation in the open eye is presumably less, and experience shows that under this pressure vitreous is sometimes lost. This complication can therefore happen on the application of pressures less than those which are produced by spasm of the lids when the most efficient retractors obtainable are in use, or which are produced by a general rise of systemic blood pressure. Under these conditions the operator may reasonably be exonerated for the loss of vitreous during an extraction, the whole blame of which may be due to the muscular action of the patient. In all operations the patient should be

instructed to avoid this muscular action, and moreover, not to strain in any way, but to lie lax, breathing regularly and deeply; and in any case where the indications point to these instructions as not likely to be carried out, a preliminary injection of novocain provides the only absolute safeguard against accident.

W. S. DUKE-ELDER.

(10) **Kyrieleis, Werner (Würzburg).—On unusual manifestations of pulsation in the vessels of the human fundus.** (**Über seltene Pulsationserscheinungen an der Gefässen der menschlichen Augenhintergrunde.**) *Arch. f. Augenheilk.*, Vol. XCVII, December, 1925.

(10) As far back as 1855 Donders established that the retinal venous pulsation is an altogether physiological occurrence in the normal eye. Although by means of Gullstrand's binocular ophthalmoscope Kümmell has been able to observe it in practically every eye examined the usual uniocular ophthalmoscopy does not reveal it. The normal venous pulsation is centripetal. In view of the numerous anastomoses of the ophthalmic vein with the veins of the face, the pulsation cannot be explained mechanically as a periodical obstruction to the passage of blood. But that under exceptional conditions venous pulsation could result from mechanical obstruction and be observed by ordinary ophthalmoscopy is shown by **Kyrieleis** in the following case:

An unconscious man suffering from comminuted fracture of the skull was examined ophthalmoscopically two days after the accident. The right eye was both externally and internally normal. The left eye showed abrasions, burns and extravasations of blood in both lids, ptosis, mydriasis and immobility of pupil and slight exophthalmos. The fundus showed slight but unmistakable pulsation of the retinal veins; it was synchronous with the radial pulse and immediately presystolic. The optic disc was sharply defined. The day after the condition was still unchanged. At the last examination which had to be done three weeks later no pulsation could be made out and exophthalmos had practically gone. The author thinks that the explanation of this phenomenon is to be sought solely in a mechanical obstruction to the outflow of blood and that, from the findings at the first examination, its true relation to the cardiac cycle, and its course, it is certain that it was not a case of pulsating exophthalmos. In all probability there was an extravasation of blood behind the globe compressing the venous outlet near the place where the central vessels leave the optic nerve and where the anastomoses with the ophthalmic vein are not yet effective.

The second unusual case is one of pulsation of a choroidal artery in a highly myopic eye which had been operated on after the method of Fukala. As the result of secondary glaucoma there was almost complete atrophy of the iris whose remnants were adherent to the cornea at the periphery. The vitreous and fundus showed very advanced myopic degenerative changes. The choroidal vessels were strikingly well seen. One choroidal artery, springing with a sharp kink from the medial border of the ring-shaped posterior staphyloma and arching upwards, showed marked pulsation. There was no pulsation whatever to be recognized in the remaining choroidal vessels. T.=55 mm. Hg (Schiötz), B.P. 90/60 (Riva-Rocci).

Although an increase of intraocular pressure closely approximating the diastolic blood pressure naturally favours the spontaneously visible pulsation, the author thinks that it cannot be the sole factor in the given case as otherwise the pulsation should have been visible in the other choroidal vessels as well. The other factor is the mechanical obstruction due to the sharp kink that the vessel makes at the border of the staphyloma where it disappears into the choroid. There is a diagram in the text to illustrate this condition.

D. V. GIRI.

(11) **Ferree, C. E., and Rand, G. (Bryn Mawr, Pa.), and Monroe, M. M. (Philadelphia).—Studies in perimetry. 1. Preliminary work on a diagnostic scale for the form field.** *Amer. Jl. of Ophthal.*, February, 1926.

(11) **Feree, Rand** and **Monroe** have investigated the fields of 200 individuals with normal fundi in order to determine the range of variation which may be expected in non-pathological cases. The ideal method of comparison would be to measure the total area of the field of vision as plotted on a standard chart. Certain practical difficulties occur in this, and an almost equally good result is obtained by taking the mean of the extent of the field in degrees in the eight cardinal directions. The investigations were made with Ferree and Rand's special perimeter using a white object, subtending a visual angle of 1°, on a black background. In general, it was found that emmetropes and hypermetropes have the wider fields and myopes, the narrower. There was only one case whose average breadth of field was less than 60°. It may therefore be stated that any field must be regarded as suspicious, if its average breadth is less than this amount. It was also found that out of 125 cases of emmetropia only six showed an average breadth of less than 64° so that 64° should be the critical figure in such cases—in myopes or presbyopes, it is about 62°. The shape of

the field is naturally of more importance in the diagnosis of pathological changes than the size, but the present paper is concerned only with the latter factor. The authors note that "the amount of contraction of field produced by a defect of refraction in a non-pathologic eye may be as great, or even greater, than that produced by an incipient pathologic condition in an emmetropic eye."

F. A. WILLIAMSON-NOBLE.

(12) **Lagleyze, M.—Double intraocular foreign body. (Doble cuerpo extraño endocular.)** *Arch. de Oftal. de Buenos Aires*, Vol. I, p. 70, December, 1925.

(12) **Lagleyze** records a case of two foreign bodies in the eye, which he considers interesting as showing the advisability of attempting removal in all such cases before it is rendered difficult or impossible by the formation of fibrous encapsuling tissue.

The patient came to the clinic with pain, photophobia, and pericorneal injection, and showed a corneal perforating scar and the formation of a hyphaema. X-rays revealed a foreign body in the eye, which was found to be magnetizable and was thus removed. The eye thereafter quietened down, but three months later he returned with a renewal of symptoms. An accidental radiographic examination (undertaken for other purposes) revealed a second foreign body which had escaped notice in the first examination, and ophthalmoscopic examination showed a yellow mass lying in the vitreous just behind the ciliary body at the extreme periphery. This was also magnetizable but all attempts with the magnet failed to remove it. As all vision had disappeared, the tension was raised, and the pain considerable, the eye was enucleated. A piece of metal was found lying deeply embedded in fibrous tissue, with which was associated a shallow detachment of the retina.

W. S. DUKE-ELDER.

(13) **Thiel, R. (Berlin).—Slit-lamp microscopy of the eye in ultra-violet light. (Ein Beitrag zur Spaltlampenmikroskopie des Auges im ultravioletten Licht.)** *Zeitschr. f. Augenheilk.*, Vol. LVIII, p. 86, 1925.

(13) **Thiel** has studied the microscopy of the living eye as seen through the slit-lamp by the aid of ultra-violet light, the appearances being rendered visible by means of fluorescence. He uses a carbon arc fitted on a slit-lamp whose optical system is of quartz, and induces fluorescence by giving the patient 2 to 3 gms. of sodium fluorescine in tea or coffee 30 or 40 minutes before the examination.

As with all red-free light the blood-vessels appear black, and around them a narrow circumscribed space is evident which is clearly fluorescent, appearing as a greenish-yellow line. This represents the perivascular lymph sheaths, and when the vessels are engorged, as in inflammatory states of the iris, this completely disappears. The fluorescine quickly finds its way into the aqueous, which fluoresces. The normal cornea shows no characteristic appearance, but folds in Descemet's membrane show up clearly in a light violet colour in the reflected fluorescent light from the lens. The lens cortex fluoresces as a pale yellow and ghostly green, while the nucleus appears yellow-grey. Shagreen of the lens is well recognized. Capsular opacities, inflammatory deposits, subcapsular vacuoles, etc., are well shown up: pigment on the anterior capsule, posterior synechiae, and the remains of past iritis appear as a milky blue, and the presence of minute opacities in the anterior cortex are much more readily detectable than by natural light and throw dark needles of shadow back into the body of the lens. Nuclear opacities show slight or no fluorescence, and therefore the opaque parts of the lens appear as blue-violet islands in the yellow-green of the fluorescent light; opacities in the anterior cortex show the same light violet reflex as the adjacent iris; opacities in the posterior cortex appear as dull reddish-grey, and fluid vacuoles and lacunae as a faint blue.

The swelling and degeneration of the lens fibres run parallel with a decrease in fluorescence, and in this way the development of a cataract can be closely followed, particularly the forms following needling and injury. For the same reason, an after-cataract does not fluoresce. A definite indication of the extent of lenticular damage after trauma is thus offered, or of the efficiency of a discission. Thiel also asserts that he has by this means substantiated the claim of Elschnig (*Klin. Monatsbl. f. Augenheilk.*, Vol. XLIX, p. 444, 1911) that there is a considerable formation of new lens fibres after an extraction due to proliferation of the epithelial remnants, and disproved the statement of Greef (*Path. Anat. d. Auges.* in Orth's. *Path. Anat.*, p. 483, 1902-06) that the whole of an after-cataract is due to incomplete removal of the cortex at the time of operation.

One of the advantages claimed for the method is that in conditions where one cannot see into the eye with ordinary light, the fluorescent light is appreciable; much information can thus be obtained regarding the inner structures in cases where the cornea is so opaque or the anterior chamber so filled with exudate that other methods of investigation are of little use.

W. S. DUKE-ELDER.

(14) **Gourfein, D. (Geneva).—Diocain in ophthalmology. (La diocaine en ophtalmologie.)** *Rev. gén. d'Ophtal.*, January, 1926.

(14) **Gourfein,** while paying tribute to the many virtues of cocain, apparently maintains that diocain is for all purposes better. He gives a list of the disadvantages of cocain namely, the effect on the corneal epithelium and on the pupil and accommodation, and its toxicity. Diocain on the other hand, gives rise to no "unpleasant sensation," is much less toxic than cocain, can be sterilized every day without losing its efficacy, produces when instilled a complete and rapid anaesthesia, and, when injected subconjunctivally, causes no hyperaemia or corneal lesions. It can be instilled in large quantities without causing trouble and without alteration of the corneal epithelium. It does not affect the accommodation nor the tension, nor does it produce mydriasis. According to the author's own statement he has studied the drug clinically only and is not speaking of its toxicity "from the physiological standpoint." He refers to the work of his "young colleagues," Koby and Lussi. Now, if readers will refer to the abstract of Koby's article on page 600 of the BRITISH JOURNAL OF OPHTHALMOLOGY for 1925, they will find certain disadvantages of diocain there stated, though they are not given as disadvantages compared with cocain but compared with holocain of which diocain is a derivative. (1) It is precipitated if the glass vessel containing it contains alkali and it has to be dissolved in boric acid; (2) instillation "up to 1 per cent." is painless, leaving it to be inferred that the drug is not, as stated by Gourfein, free of painful effect when used in strengths higher than 1 per cent. Yet Gourfein recommends 2 to 5 per cent. solutions. (3) It is evidently fairly toxic since the author suggests that for repeated instillations the strength should not exceed two to three *per thousand*. There are certainly points of difference in the views of Gourfein and of Koby, but it is to be noted that Gourfein emphatically states that clinically he has not noticed the least toxic effect either in children or adults among over 300 cases in which the drug was either instilled or injected.

ERNEST THOMSON.

(15) **Meller, J.—Spontaneous bleeding in atrophic eyes. (Ueber spontane Blutungen in atrophischen Augen.)** *Zeitschr. f. Augenheilk.*, Vol. LVII, p. 131, November, 1925.

(15) In a long and detailed paper illustrated by pertinent cases, **Meller** discusses the question of the aetiology and effects of the spontaneous haemorrhages that sometimes occur in atrophic eyes.

He differentiates three types:

(1) Haemorrhages from the organized tissue covering of the ciliary body and vitreous which is formed during the course of the primary inflammatory disease (*Schwartenblutungen*). The new vessels in this tissue are easily liable to be ruptured, either from the mere shrinkage of the tissue itself, dragging upon them or occluding them locally, or from various trifling causes (traumatic, etc.), such as would leave the normal eye unaffected. (2) Spontaneous sub-choroidal haemorrhages, which occur usually in eyes in which the primary trouble involved a prolonged state of hypertension. The main element in the occurrence of these is suggested as a weakening of the vorticose veins and the choroidal vessels supplying them by the mechanical dilatation of the eyeball during the period of raised tension and its shrinkage after that tension has died down. These two types may occur simultaneously and independently of each other. (3) Haemorrhages in the tissues of the eye itself; this occurs more rarely. In the paper two illustrative cases are given, in one of which such a haemorrhage occurred in the pupillary part of the iris, and in the other it was due to a tear in the retina caused by contraction of organized exudative tissue (*schwartenzug*).

Two effects of these haemorrhages are discussed:

(1) They produce a rise in pressure—a purely volume effect. This is usually of little consequence, the shrunken eyes often remaining still more hypotonic than normal after the haemorrhage. (2) An inflammation which is often set up after the haemorrhage, independent of the inflammatory infiltrations inside the eye, is of more importance. This is probably responsible for the sudden inflammatory attacks which often occur in atrophic eyes which have long been quiet, an occurrence which often necessitates enucleation. The new haemorrhages from the old inflammatory tissue produce an inflammatory reaction with a histologically recognizable infiltration round about it, due to changes taking place in the composition of the extravasated blood after it has remained for some time. A repetition of this bleeding, with its consequent reaction, brings on a sudden severe disturbance of an anaphylactic nature. This process occurring in the sub-choroidal vessels Meller regards as being the cause of "pseudo-sympathetic choroiditis"; and he describes several cases wherein a similar delayed and severe "sympathetic" inflammation in such atrophic eyes could be definitely associated with recurrent reaction-producing haemorrhages. The author considers that these facts should weigh in the consideration of the advisability of enucleating eyes of this atrophic type.

W. S. DUKE-ELDER.

BOOK NOTICES

The Ophthalmic Year Book, Vol. XXII. Containing Bibliographies, Digests and Indexes of the literature of Ophthalmology for the year 1925. Edited by WILLIAM CRISP, with the assistance of several collaborators. Pp. 313. The Ophthalmic Publishing Co., Chicago, 1926.

The present volume of this valuable year book brings the references up to those included in the "current literature" lists of the *American Journal of Ophthalmology* for December, 1925. These lists furnish practically the entire basis for the bibliographies, lists, and digests which constitute the year book. The general arrangement of the book remains as in past volumes, but some endeavour has been made to economise space by the omission of references to work of little or no permanent value. The ophthalmic world owes a debt of gratitude to the writers of this indispensable book for their painstaking and often very dreary work.

Stilling's pseudo-isochromatische Tafeln zur Prüfung des Farbensinnes. Heransgegeben von Geheimrat PROF. DR. E. HERTEL. 16th Edition. Leipzig: Georg Thieme, 1926. Price 18 marks.

We welcome a new edition of these well-known "pseudo-isochromatic" tests for colour blindness. Their utility has been fully proved, though like all other tests for colour blindness they have their limitations.

Tafeln mit Umschlagfarben zum Nachweis von relativer Rot-und Grünsichtigkeit. von Dr. Med. ERNST WÖLFFLIN. Basel: Leipzig: Georg Thieme, 1926, Price 5 marks 70.

These tests are a refinement of Stilling's tests. Some years ago Ishihara added to Stilling's tests some plates in which the designs were easily discriminated by the colour blind but not by those of normal sight. In Wölfflin's plates the hues are similarly visible to the colour blind and invisible to the normal sighted by daylight. They can, however, be discerned by the normal under artificial illumination. The difficulty, already very great in Stilling's tests, of obtaining precise colours in the lithographic plates must have been greatly increased in these. The extreme delicacy of the tests may be expected considerably to limit their practical utility.

Hilger's Spectrometers. ADAM HILGER, 24, Rochester Place, N.W.1.

We have received a list, with descriptive details, of Hilger's Spectrometers. These instruments are unsurpassed in accuracy

and perfection of workmanship. They afford the means of analysis of the spectrum from the extreme ultra-violet (1,850 Å) to the extreme infra-red (100,000 Å). Their prices are mercifully withheld.

NOTES

Writing in the Cloister

To those who have wondered how the early monastic chroniclers were able to do their literary work in the ill-lit monastic buildings the following extract from Ordericus Vitalis, *Hist. Eccl. Lib. XIII* may be of interest.

"Behold, worn out with age and infirmity, I desire to end my work, and for many reasons prudence requires it. For I am now (1141) passing the 67th year of my age in the worship of my Lord Jesus Christ."

Ibid. Lib. IV. "But now I am benumbed with the winter frosts and must turn to other duties: wherefore I am resolved here to lay down my pen and end this fourth book. When the soft air of sweet spring shall return, then I will turn again to those matters which I have omitted or have treated too briefly."

Ordericus was a monk of St. Evroul in Normandy. These extracts show him, an old man, still writing at nearly 70 years of age. Spectacles were unknown at this time, and his literary efforts seem to have been bounded by numbed fingers rather than by visual disability. Perhaps, after all he was a myope.

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Appointments

MR. R. C. DAVENPORT has been appointed Assistant Surgeon to the Central London Ophthalmic Hospital.

DR. WALTER KIEP has been appointed Ophthalmic Surgeon to the Victoria Hospital, Keighley, and DR. W. D. HAMILTON Assistant Ophthalmic Surgeon to the same institution.

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Royal London Ophthalmic Hospital Dinner

THE dinner of past and present students of the Royal London Ophthalmic Hospital will take place on Thursday, February 10, 1927. Mr. Percy Flemming will be in the chair.

The Annual Congress of the Ophthalmological Society of the U.K.

THE Annual Congress will be held in Liverpool on Thursday, Friday, and Saturday, April 28, 29, and 30, 1927.

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Decennial Index

A COMPREHENSIVE Index of the Original Articles contained in Vols. I to X of the BRITISH JOURNAL OF OPHTHALMOLOGY will be issued with the January number.

FUTURE ARRANGEMENTS

1926

December 3.—North of England Ophthalmological Society, at Bradford.

December 10.—Royal Society of Medicine, Section of Ophthalmology (Clinical).

1927

January 14.—Royal Society of Medicine, Section of Ophthalmology.

February 4.—North of England Ophthalmological Society, at Liverpool.

February 10.—Royal London Ophthalmic Hospital Dinner.

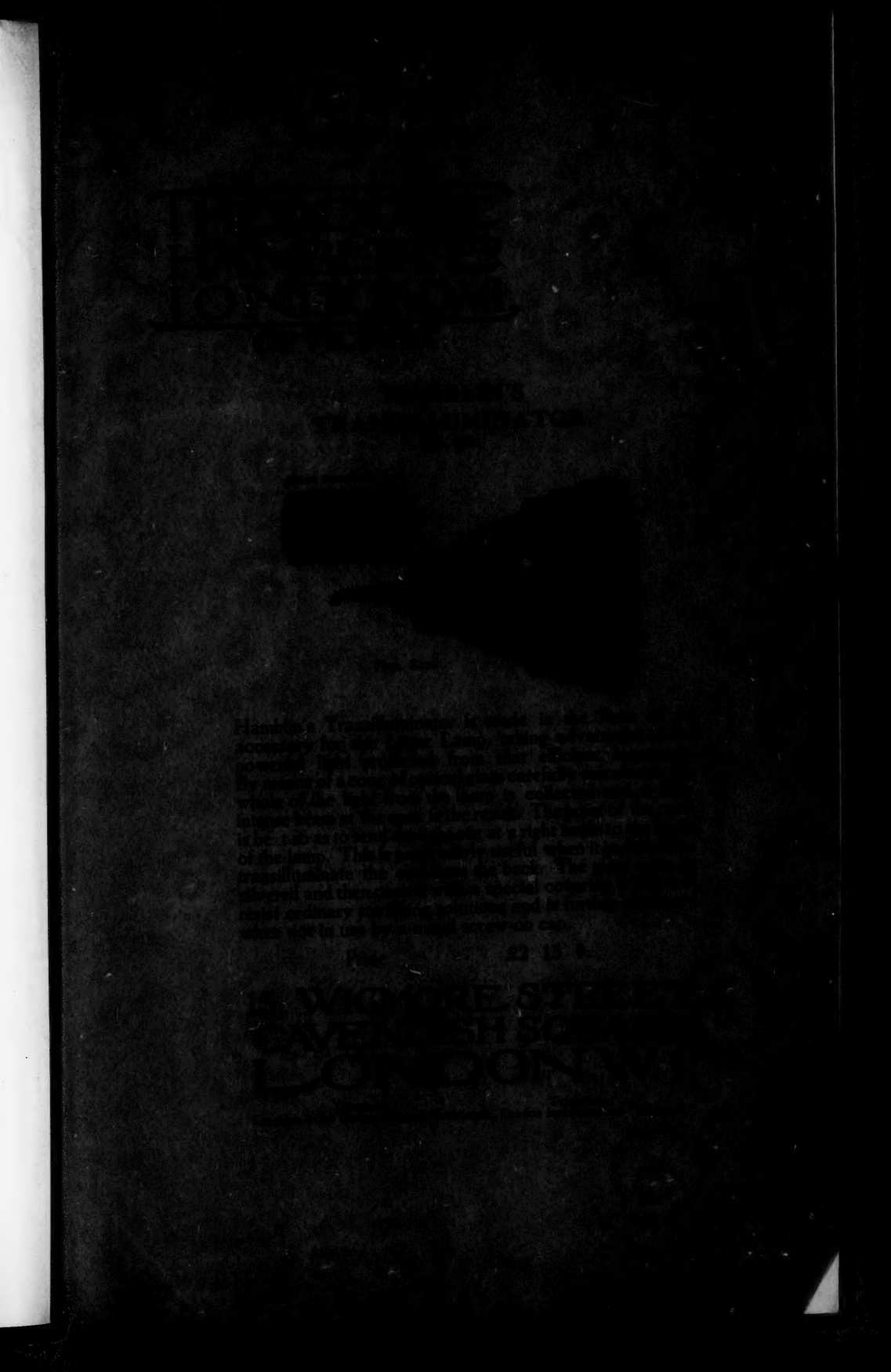
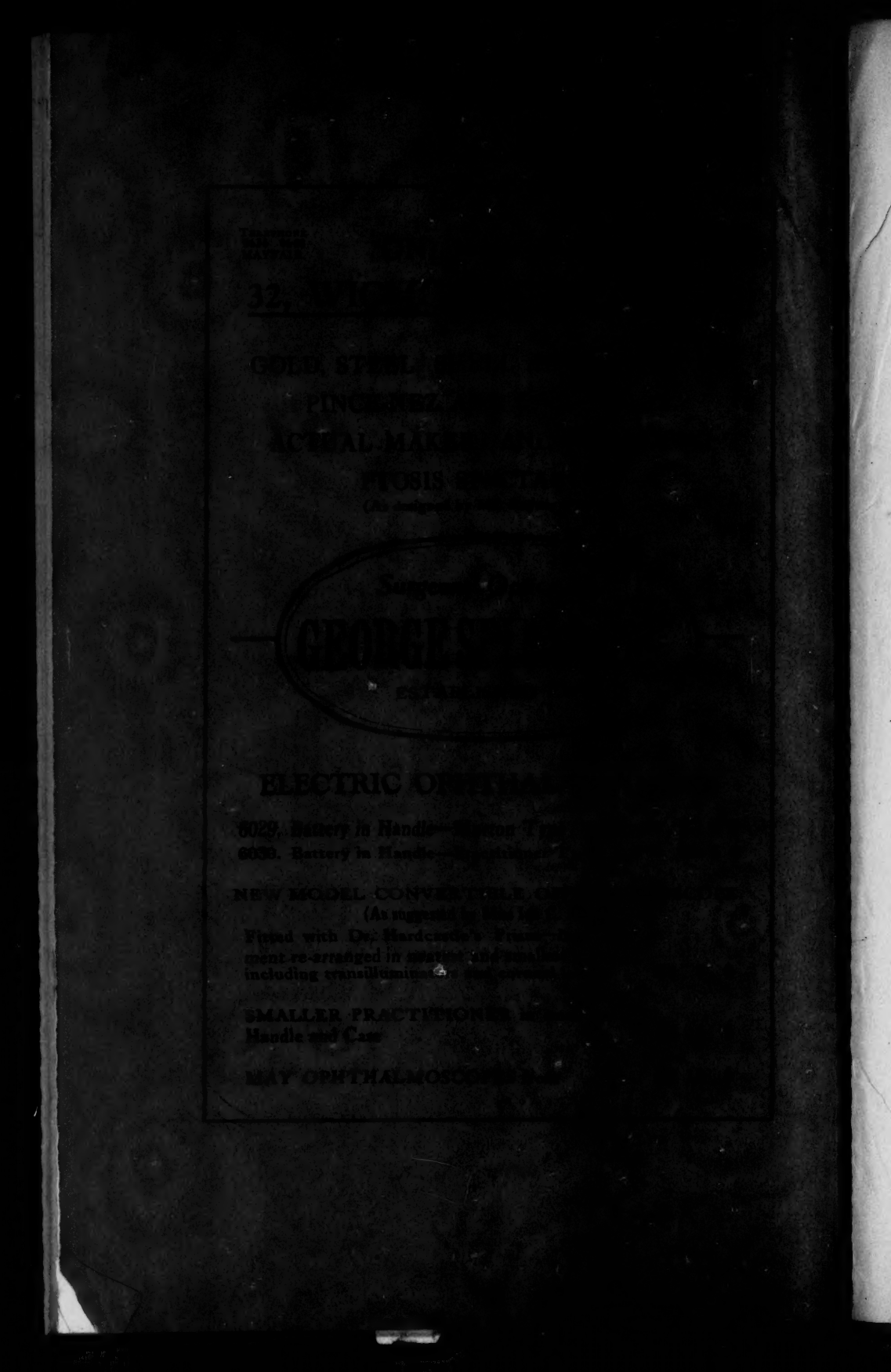
February 11.—Royal Society of Medicine, Section of Ophthalmology.

March 4.—North of England Ophthalmological Society, at Sheffield.

March 11.—Royal Society of Medicine, Section of Ophthalmology (Clinical).

April 1.—North of England Ophthalmological Society, at Newcastle-on-Tyne.

April 28-30.—Annual Congress of the Ophthalmological Society of the United Kingdom.

May 13.—Royal Society of Medicine, Section of Ophthalmology.

June 10.—Royal Society of Medicine, Section of Ophthalmology (Annual).